

NEW (5th) EDITION

# *DeLee's Obstetrics*

Both text and illustrations received careful revision in the preparation of this Fifth Edition. ¶ Dr DeLee rewrote completely the chapters on the Treatment of Hyperemesis, Eclampsia, Abruptio Placentæ, Placenta Prævia, Ruptura Uteri, Postpartum Hemorrhage, Breech Presentation, and the Operation of Forceps ¶ The chapter on Contracted Pelvis reflects the latest simplified methods of treatment ¶ The chapter on Forceps is much enlarged and contains new illustrations ¶ The illustrations for the new, low, or cervical cesarean section—laparotrachelotomy—have been improved and increased in number to make the operation more easily learned ¶ The operation of Gottschalk-Portes, temporary explanation of the infected uterus, has been briefly described ¶ In this Edition, too, he has emphasized the causes and prevention of maternal mortality

You will pronounce this book the most elaborate, the most superbly illustrated, the most instructive work on Obstetrics you have ever seen

You will find the text extremely practical throughout, Dr DeLee's aim having been to produce a book that would meet fully every need of the practitioner as well as the obstetrician For this reason *Dagnosis* is featured Regarding *Treatment* You get here the very latest advances in this field, and you can rest assured every method of treatment, every step in operative technic, is just right

By J. STEWART B. DELEE, A. M., M. D. Professor of Obstetrics at the Northwestern University, Chicago, Ill. Revised Edition of 1917, 200 pages, with 2125 illustrations, 8vo. in cloth. Price \$12.00 net.

W. B. SAUNDERS CO., Philadelphia and London

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These lesions passed through the usual stages of erythema, vesication, and scaling. There were somewhat milder lesions on the tops of the feet.

Although the pellagrous lesions that developed first have greatly improved, there are two noteworthy conditions to which I wish to direct attention. One of these is the thickened, hard, and very rough skin over the nose, especially the sides, the chin, and, to a less extent, the forehead. No erythema or exfoliation occurred, and there was little or no change of color.

I have often noticed this condition of the skin of the face, particularly in severe cases of pellagra in negro women. The skin becomes thick and hard. When asked about it, the patient may note a feeling of numbness and stiffness. Hardened sebaceous material protrudes from the sebaceous glands where these are most numerous and active, as on the sides of the nose, and on the chin of "greasy-faced" negroes, giving to the skin a very rough grater-like feel.

The other skin lesions to which attention is directed consist of severe lesions on the points of the elbows. The lesions on the hands, neck, and feet ran the usual course for such lesions, but a few days after the patient was admitted to the hospital she developed a large severe lesion on the right elbow from which the skin slipped off, leaving it raw and bleeding, and a smaller, less severe, very dark lesion on the left elbow. These lesions on the elbows developed nearly a month after the first lesions on the hands began and only after the patient's general condition became so bad that she was confined to bed and her mental condition became so poor that she became awkward and did not exercise the usual care against abuse of herself in moving around in bed, especially in getting up and down. It often occurs that pellagra patients develop lesions on the elbows as soon as they are confined to bed. A large proportion of the acute very active cases develop such lesions especially those whose nursing and care are such as to allow them to rise and rest upon their elbows in bed.

The lesions in pellagra are caused by physical forces or chemical action on the skin in persons whose skin has lost its

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no lesions whatever would have developed? I say there could not be, nor could there be any reasonable doubt that if the band had not been protected the lesion would have developed there as it did above and below on the same finger and on corresponding areas on the other fingers

It is quite likely that exposure to sunshine of certain quality or intensity that may not now be understood may be most effective in producing the lesions on the skin of a person who has the general disease at the stage which renders him susceptible

Although exposure to sunshine in some way is the exciting cause of the lesions in most cases, there are several other things that we know of that may cause them and, no doubt, others that we do not know of. Exposure to heat will cause them, as in the case of a woman whom I once saw who developed severe lesions chiefly on the lower or ulnar side of the forearms following cooking and working for hours over a hot stove at the time she was already sick, probably from the early general effect of pellagra

Such things as pressure and trauma to the skin may cause the development of lesions as occur on the elbows of patients who are confined to bed, especially if there is more or less dementia, as in the case of our negro woman. In most very acute fulminating cases lesions develop on parts where there is pressure, such as over the heels or the bony prominences on the back, shoulders, etc

Irritating chemicals may cause lesions to develop. Strong ointments containing such agents as carbolic acid, ichthyol, and iodine, when applied to pellagrous lesions, not only make them much worse, but tend to make them spread as far as the ointment is applied beyond the lesion. I have seen lesions under the pendulous breasts of a fat woman caused by irritating, sour, unclean perspiration. The same woman had typical lesions on her hands on which the diagnosis of pellagra was based

The mucous membranes are susceptible to irritating influences in much the same way as the skin. The vaginitis and vulvitis so frequently occurring in women with pellagra result from the effect of irritating acrid vaginal discharges, against which the tissues of a normal person are more resistant

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second case of pellagra that I have any recollection of ever seeing. This was some two years before pellagra was first diagnosed in New Orleans, and, as I recall the case, it was quite typical and presented a clear picture of the disease. The patient was taken to a leading dermatologist, and to a leading psychiatrist, but neither of them thought of pellagra, nor did I.

Some three years later the physician himself began to have rather vague ill health, including especially digestive disturbances, "nervousness," insomnia, hallucinations, weakness, and some loss of weight. He was troubled with sore tongue which he attributed to chewing tobacco. This condition continued for more than a year, better at times, and worse at others.

Following a period of three or four weeks during which he was confined to bed, or to the house most of the time, he got what he first thought was sunburn on his hands as a result of exposure to bright sunshine while riding horseback. This developed to typical pellagrous lesions upon which we were now able to make the diagnosis with certainty, since we had had our attention called to the disease, and had learned to recognize it.

After a few months during which the patient had a rather "rocky time," he went to Colorado, where he slowly improved so that he was able to return to his home in Mississippi, where he is still living and practising medicine, never having had pellagrous lesions again.

The feature in this case to which I wish to direct attention is the evident existence of the general disease pellagra for more than a year before skin lesions developed on which the diagnosis could be made. The necessary combination of the requisite stage or period of the disease with the requisite exciting cause had not existed previously.

The question may be asked why exposure to sunshine had not previously brought out the lesions. I do not wish to convey the idea that exposure to sunshine produces lesions in pellagra patients at any time that it occurs. It is only at certain times, under certain circumstances not now understood, that it will produce them.

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*Present Illness*—Seemingly normal baby in every way Patient born three months after death of father and mother had to go to work and not able to give him the attention he needed, thinks he may have talked if he had been taught sooner Said “daddy” at age of two years, but did not talk until four years of age Weight only 7 pounds at birth Had tonsils and adenoids removed by Dr Lokey at four years, and soon after this began



Fig 192 —A case of dystrophia adiposogenitalis

to gain weight and is especially fat around the waist When weighed here in office weight is 96 pounds Height,  $51\frac{1}{2}$  inches Patient nervous and restless Squints and blinks eyes frequently Stammers ever since he began to talk Got zero in everything in school Cannot promote him Knows his letters pretty well

Mother says patient was shriveled looking from three months to four years of age Mother did not have enough milk

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11 Normal

12 Normal movements No tremor or atrophy

*Taste*—Rt ant Acid +, sweet +, bitter +, salt +

Lt ant Acid +, sweet +, bitter +, salt +

Rt post Acid +, sweet +, bitter +, salt +

Lt post Acid +, sweet +, bitter +, salt +

Speech Very difficult to understand anything he says  
Seems to understand what is said to him Stammers

Motor Abnormal muscular movements, blinking of eyelids constantly

Muscle strength Easily fatigued

Paralysis none

Posture normal

Tonus normal

Co-ordination Gait normal except feet a little far apart

Romberg absent Finger-nose normal Finger-finger normal

Trophic Excessive weight for age and height

Reflexes, deep Rt biceps +, triceps +, radial +, ulnar +, patel +, Achil +

Left biceps +, triceps +, radial +, ulnar +, patel +, Achil +

Reflexes, superficial Rt up ab +, l ab +, crem 0, plan +, Babin 0

Lt up ab +, l ab +, crem 0, plan +, Babin 0

Cavernous reflex absent

Sensation Touch +, pain +, tem +

Nutrition Weight 96 pounds Height 51½ inches

Vesical functions Wets bed every night and wet clothes often till a month ago

Rectal functions Did not control bowels till two months ago

Fever 99 2° F

Head mesocephalic, prominence of left cerebellar region, turns red when he gets hot

Ears Frequent earache and discharge since one year of age, better since tonsils were removed

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Small postnasal space    Left antrum cloudy    Fundi negative  
 Vision 20/20 without glasses and 20/15 with glasses "

Blood chemistry	Mg per 100 c c of blood
Sugar	100 0
Non-protein nitrogen	21 3
Urea nitrogen (estimated)	15 0
Creatinin	1 5
Chlorids	379 5
Calcium	14 0

### Spinal fluid

Cell count	5 per cu mm
Globulin	Negative
Sugar	Negative
Wassermann	Negative
Basal metabolic test	(-20)
Blood sugar	133 3 mg per 100 c c blood

Attempts to get carbohydrate tolerance test failed because patient vomited each time it was attempted

Six weeks later carbohydrate tolerance test shows

	Mg per 100 c c of blood
Blood sugar before glucose	95
$\frac{1}{2}$ hour after	143
1 hour after	87
2 hours after	100
3 hours after	87

During the following two years the patient improved very much in his general condition, and practically ceased wetting the bed, and his habit spasm disappeared. He continued to gain in weight, however, and reached  $131\frac{1}{4}$  pounds twenty-three months after the first note at the age of ten years and two months.

His mother was quite certain that before his operation—having his tonsils and adenoids removed—he was skinny and underweight, and immediately after the operation he began to gain weight rapidly, and finally reached a very abnormal weight for his age. I think it probable that in the removal of his adenoids a pharyngeal pituitary body was also removed and that this produced his dyscrasia.

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combination has recurred several times since, always after severe muscular effort, and has always cleared up in a few days

In childhood the patient had mumps, whooping-cough, measles, malaria, and pneumonia. There were frequent attacks of tonsillitis until 1919 when his tonsils were removed. He has been married for five years, his wife is well and has had two healthy children. The patient used a moderate number of cigarettes and drank coffee daily.

On examination the patient was a young man lying quietly without dyspnea or orthopnea. There was a decided malar flush, the fingers were not clubbed, and there was no fever.

The cardiac dullness extended 4 cm. to the right and 10 cm. to the left. The apex-beat was localized and tapping and was preceded by a definite short thrill. The rhythm was regular and the rate 70. On auscultation there was a loud snapping first sound preceded by a long rumbling diastolic murmur with a presystolic crescendo. No systolic murmur was heard at the apex and no murmurs were heard at the base. The pulmonic second sound was loud and ringing.

There was no evidence of peripheral congestion in the neck veins, no râles in the lungs, and no edema anywhere. The liver and spleen were not enlarged. Change in position caused a marked acceleration of the heart rate.

There were no abnormalities in urine or blood. The electrocardiogram showed large P waves and a right ventricular preponderance.

This patient had the classical signs of a severe grade of mitral stenosis. His symptoms are of interest in that they date far back into his youth and that in general, they are precisely the symptoms brought on in a healthy man by vigorous exertion. Their significance consists in the fact that shortness of breath (which is the chief symptom) occurs after much less exertion than is required to produce it in even a moderately healthy man. Now the conception of a "cardiac reserve" is a familiar one. It implies that in response to a need for a greater oxygen supply, such as exists during exercise, the normal heart can increase the amount of blood it pumps to the tissues to several times the amount needed by a resting body. But every heart has its limit, and when that limit is reached the owner of the heart will suffer from shortness of breath. Thus the amount of blood the heart can pump per minute is one of the factors which determine the degree of muscular exertion possible for a given individual.

This patient has an extremely limited exercise tolerance, he has only a small cardiac reserve. This limitation is apparently

combination has recurred several times since, always after severe muscular effort, and has always cleared up in a few days

In childhood the patient had mumps, whooping-cough, measles, malaria, and pneumonia. There were frequent attacks of tonsillitis until 1919 when his tonsils were removed. He has been married for five years, his wife is well and has had two healthy children. The patient used a moderate number of cigarettes and drank coffee daily.

On examination the patient was a young man lying quietly without dyspnea or orthopnea. There was a decided malar flush, the fingers were not clubbed, and there was no fever.

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electrocardiogram showed an auricular rate of 300 and a ventricular rate of 150, with the characteristic curve of auricular flutter

The patient was given 16 gm of digitalis in three days. There were periods of complete irregularity of the pulse on the second and third days. These were shown by the electrocardiogram to be due to an irregular block and not to any change in the auricular mechanism. On the fifth day there was an abrupt change to a normal sinus rhythm with a rate of 75. The patient was conscious of the change of rhythm, and immediately felt a sense of relief, of comfort, and of increased strength. The dyspnea disappeared after the change in rhythm and rate, the blood-pressure was then found to be 120/72

In this patient we have an old man with some arteriosclerosis and a profound disturbance of the rhythm of the heart, leading to a constant rate of about twice the normal. The reversion of the rhythm to normal which was accomplished by the administration of digitalis and which was accompanied by a decrease in rate, resulted in prompt relief from the weakness and shortness of breath from which he suffered. His color also improved, and the pulse pressure more than doubled, as may be seen in Fig 193

The low blood-pressure, the cyanosis, and the weakness suggest that during the period of abnormal rhythm this patient was in a condition akin to surgical shock. In this condition the amount of blood put out by the heart has been shown experimentally to be diminished. The assumption of decreased output, as well as decreased blood-pressure, explains his symptoms satisfactorily as being due to an actual diminution in the oxygen supply to the tissues. They are symptoms of oxygen lack. It is important to note that digitalis was given to change the rhythm responsible for the failure and not to treat the failure itself, because in the shock type of failure digitalis probably does harm rather than good. As is usual in the type of circulatory failure exemplified by this patient he showed no edema of either lungs or periphery.

**Case III.**—The third patient was a colored woman forty years old, who entered the hospital complaining of attacks of a smothering sensation at night. She dated the onset of the symptom to about a year before admission when she noticed that after she had been in bed for an hour or two she would often awake feeling short of breath. This was relieved promptly by sitting up in bed. Later she began to have the same discomfort after eating a large meal or walking briskly up stairs. With these attacks there were sometimes cough and frothy sputum.

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albumin and many granular and hyaline casts The blood contained 3,900 000 red cells, 8000 leukocytes, and 60 per cent hemoglobin by the Sahli method There were 36 mg of non-protein nitrogen per 100 c c of blood The  $\text{CO}_2$  combining power of the blood was 68 volumes per cent The phenolsulphone phthalein excretion was 40 per cent in two hours The Wassermann reaction was negative

After eight days of rest in bed the patient's blood-pressure was 240/135 At the end of this period at 9 15 one evening she had an attack of severe dyspnea during which she sat up in bed, breathed rapidly, and was in great distress She coughed frequently, raising small amounts of foamy sputum Examination of the chest showed many râles varying from fine moist ones to sonorous ones and most numerous over the lower lobes Shortly after the administration of 15 mg of morphin sulphate the paroxysm was relieved

We see in this patient the victim of severe vascular disease, and very marked hypertension The first and chief symptom of her heart-failure is paroxysmal pulmonary edema, usually occurring either at night or after exertion The blood-pressure was not further elevated during the attack in this patient, but a study of a series of similar cases reveals that it is often higher than usual In this case it may have been elevated above its usual height just prior to the attack The paroxysm passed off after the administration of morphin There is no evidence in the blood-pressure, the full pulse, the color, and the warm extremities of these patients, of any diminution of cardiac output, nor is it easy to see why a diminution of output should lead to a rapid accumulation of edema in the lungs only

It is necessary here to introduce another factor into our discussion of heart-failure It is obvious that to maintain normal circulatory mechanics there should be perfect co-ordination between the right and left ventricles, and that the two ventricles should expel exactly the same amount of blood with each beat, otherwise blood would rapidly accumulate either in the lungs, if the right ventricle puts out more than the left, or in the systemic circulation if the opposite condition holds Normally the co-operation is perfect, each ventricle adapts itself instantly to receive and dispose of the amount of blood sent to it by the other When the heart is damaged, however, especially when the damage is more extensive in one ventricle or when an excessive load is imposed on one ventricle, it is to be expected that the ability

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changes in voice or breath sounds, but a few scattered râles at the bases of both lungs

The area of cardiac dullness extended 12 cm to the left and 6 cm to the right of the midsternal line. The supracardiac dullness was 7 cm in diameter. The heart rate was extremely rapid and the rhythm totally irregular. The rate at the apex was 180, and at the radial 100, a pulse deficit of 80. The first sound at the apex had a snapping quality, but no murmurs were heard at any area. The blood-pressure (taking the beats of maximum pressure) was 140/90.

The abdominal wall was edematous, but it was still possible to palpate a smooth, slightly tender liver edge 6 cm below the costal margin. There was shifting dullness in the flanks and an easily demonstrable fluid wave.

The skin of the legs was tense and glossy. There was a slight, fine tremor of the extended fingers.

The urine had a specific gravity of 1.018, and contained albumin but no sugar. The sediment contained a few white blood cells and no casts.

The blood contained 3,700,000 red blood-cells and 10,800 white blood cells (79 per cent of which were polymorphonuclears) per cubic millimeter, and 70 per cent of the expected hemoglobin. The Wassermann and Kahn reactions were strongly positive. The non-protein nitrogen was 25 mg per 100 c c. The phenolsulphonephthalein excretion was 45 per cent in two hours.

The electrocardiogram showed a ventricular rate of 180. There were no regularly spaced P waves, but many irregular fibrillary waves.

A teleroentgenogram of the heart showed an arch 7 cm in diameter. The left border of the heart was 11.5 cm and the right border 6.5 cm from the midline.

The patient was put at absolute rest in bed, the daily intake of fluids was limited to 1200 c c, and the administration of digitalis was begun. In the first twenty-four hours she received 1.2 gm of digitalis leaf. At the end of this period, the apical rate had fallen to 130 and the patient was much more comfortable. She received on the second day 0.6 gm of digitalis leaf, and the same amount on the third day. When 2.4 gms had been given in three days the apical rate was 90, there was no pulse deficit, and the patient's weight had fallen from 216 to 190 pounds. She was then placed on a daily dose of digitalis of 0.1 gm.

On the sixth day after admission, with pulse 90, the basal metabolic rate was determined and found to be 69 per cent above the calculated normal. This was repeated three days later, and was 63 per cent above normal. At this time the patient weighed 175 pounds.

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At this time the patient felt entirely well She had no shortness of breath and was free of palpitation for the first time in years Her weight at this time was approximately 25 pounds above the low point She was discharged from the hospital ward to the syphilis clinic where she began a thorough course of anti-syphilitic treatment

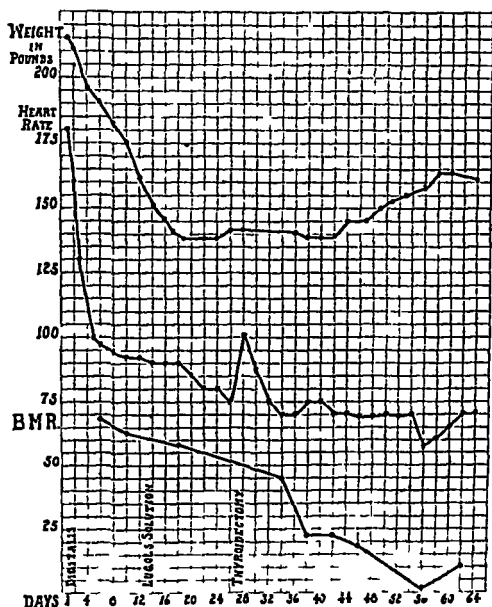


Fig 194 —The weight of the patient during her stay in the hospital, charted against heart rate and basal metabolic rate

She was last seen in July, 1928 At that time she considered herself well, was doing her housework without breathlessness, nervousness, or fatigue, showed no edema and weighed 203 pounds

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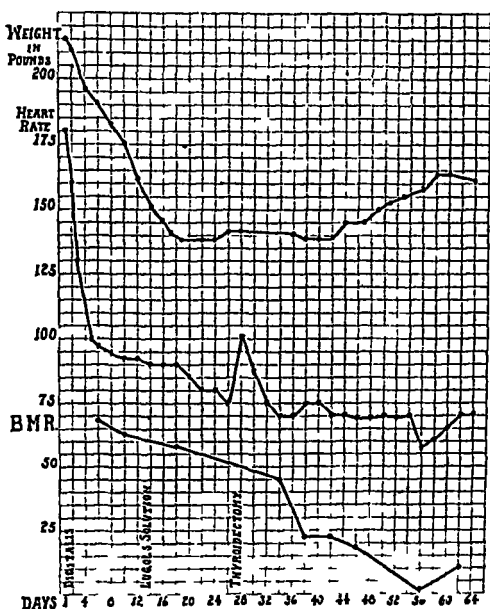


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The changes in weight which constitute for us this patient's chief interest are shown graphically in Figs 194 and 195. Figure 194 shows the fluctuations in weight during the patient's stay in the hospital charted against the fluctuations in pulse rate and basal metabolic rate. The initial enormous drop in weight is seen to follow the control of the heart rate by digitalis and to occur before the major changes in the metabolic rate. The gain in weight, on the other hand, is seen to occur after the fall in metabolic rate induced by thyroidectomy. Figure 195 is an

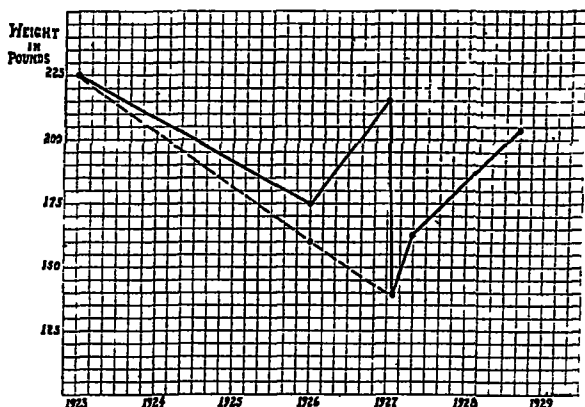


Fig 195—The variations in the weight of the patient over a period of five years

attempt to indicate roughly the changes in weight since the beginning of the patient's illness four years before admission. It consists of two curves, one in which the actual body weights are charted, and a second, represented by the dotted line, in which the attempt is made to estimate the weight of the body without edema. In 1926, for example, a year before admission to the hospital the patient weighed 175 pounds. At this time she had some edema and since visible edema implies the presence of about 15 pounds of fluid at the least, that amount was subtracted from the gross weight. This procedure makes possible the construction of a weight curve the changes in which are due chiefly to the

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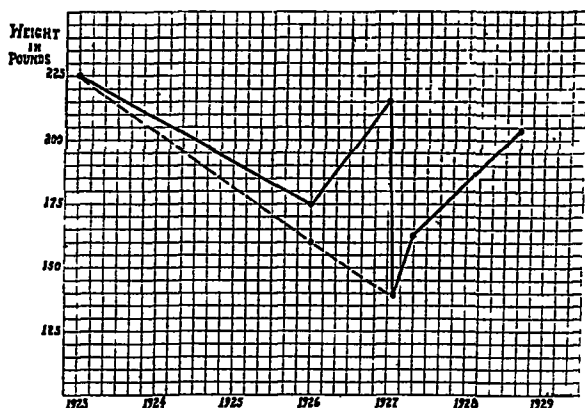


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the other hand many types of patients undereat. It is easy to see that people who cannot get enough food must suffer from starvation, and it is equally important to recognize that patients whose intake of food is reduced because of disease also suffer from starvation. Starvation in patients may be due to actual failure to ingest enough food, as in anorexia of all sorts, and in obstruction in the esophagus, it may be due to failure to absorb ingested food, as in persistent vomiting or constant diarrhea, or it may be due to disturbance of metabolism after the food is absorbed, as in diabetes mellitus.

In most cases more than one factor is operative. Thus in infections there is not only the elevated metabolism due to fever, but also the disturbance of appetite associated with the malaise. Similarly the loss of weight which is so important a sign of malignant disease may be due either to disturbances in digestion or absorption from the new growth itself, to reduction in the desire for food, or to increase in the metabolic rate.

The second group of causes of changes in weight has to do with variations in the amount of fluid in the body. Acute diarrhea may cause a loss of body fluid, but the usual way in which body fluid first alters body weight is by its accumulation in the body. Accumulation of fluid may occur as the edema of heart-failure or nephritis, as the ascites of portal obstruction, or as an exudate poured out in a serous cavity in response to some inflammatory change. This accumulation causes a gradual or sudden increase in weight. Removal of the edema by diuresis, tapping, or other procedure causes a converse change. Generalized edema, by its effects both upon the weight and upon the appearance of the patient, often masks a considerable loss of body tissue. Under these circumstances the loss of the edema will reveal an unexpected degree of emaciation. Most diseases which are capable of causing generalized edema can so interfere with the normal metabolic processes in the body as to cause also a loss of body substance. In a given case several factors may be present, some tending to increase and some to diminish the total weight of the body.

In addition to these more usual mechanisms of loss and gain,

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In this connection I might mention that tuberculosis and high blood pressure are not common bed-fellows. In fact most tuberculosis cases have a low blood-pressure even in the early stages before debilitating effects of the disease have become prominent. One observer reports a group of 162 tuberculosis cases showing an average blood-pressure of 106/75, as compared with a control group of non-tuberculous individuals whose average blood pressure was 120/80. Low pressure seems to be part of the strumous individual.

This man's appearance, therefore, suggested to me that he was of the type who had little energy and a poor supply of reserve strength, rather than the florid, energetic, driving type who so much more frequently show accelerated circulation and high blood-pressure. So, without prejudging him, I must confess that I would not have been surprised to find some early evidence of tuberculosis. His lungs were perfectly normal, however, and I was on the contrary somewhat surprised to find his blood-pressure 160/80 with a pulse rate of 100 per minute. On further examination I found no sign whatever of any organic disease. His circulatory system especially was free from definite signs of disease, the heart showed no enlargement, the palpable peripheral and retinal blood-vessels were not thickened or narrowed or tortuous. His urine was negative, the specific gravity showed normal ability to concentrate, he had no nocturia, denied headaches, and, in fact, had had no illness of any consequence in his lifetime.

While examining his heart, however, I was impressed by the fact that while it was perfectly normal in size, it was a little rapid and contracted with more force than usual, with changes of rate and force. I do not mean that the rhythm became intermittent, I mean that there was a change of rhythm rather of the sinus type. At times the rate would speed up and systoles were strong and loud, but in a few seconds would quiet down and assume a more nearly normal rate and force. This occurred from time to time, and I also began to notice fluctuations in his blood-pressure, at times it registered as high as 185/100 while a few minutes later it would drop 10 to 20 mm both systolic and diastolic. At the same time the rate and force of his heart would diminish. He was manifestly a little nervous and showed fine tremor of his fingers. But you will note that his thyroid is not visible or palpably enlarged, and he does not impress one at all as a hyperthyroid case, and I might say also, that a recent basal metabolic observation was normal. He seemed relieved and anxious to be over with the examination.

This is undoubtedly a hypertension of nervous origin. After seeing him a number of times, when I took ample time to talk to him in a reassuring manner, I inspired confidence in him and explained to him that he had no real organic disease and that his blood-pressure was not a serious condition, but was merely elevated as a result of the nervous excitement while under examination. I did not hesitate to tell him that if I could in some way take his blood-pressure at night when he was soundly sleep

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nervous circulatory phenomena. I call attention to this type of case because in the milder forms it is often difficult to recognize at once as being a functional and transient condition, and many are made worse by being branded as high blood-pressure cases. A vicious circle ensues: their blood-pressure rises because of nervous causes, and their nervous equilibrium is further disturbed by the thought and fear of high blood-pressure.

**Case II.**—This next case is a lady who has been a patient of mine for ten years. She is now fifty-six years old. Her father died of apoplexy, her mother of heart trouble at fifty-six, one brother died of Bright's disease, one sister died suddenly in bed, four sisters are living and healthy, except one, who has high blood-pressure. Three brothers are living and healthy.

When I first saw her in 1918 she told me she had never been sick, she had not consulted a doctor in twenty-five years. Suddenly she had epistaxis which continued intermittently for a week. Her blood-pressure was found to be 200 at this time, dropping to 130 with the hemorrhages. She has been under my observation continuously ever since, and she is just as well today as she was ten years ago, except perhaps at times when nervousness and apprehension over her blood-pressure take hold of her and almost make her a nervous wreck. Her physical findings have varied little in ten years. She must have had a high blood-pressure for years before the epistaxis, because her heart showed left ventricular hypertrophy when first seen. She is neither stout nor thin, nor pale nor florid, her general appearance is of good health. Her eye-grounds show no findings of note, the vessels are neither tortuous nor sclerotic, her lungs and abdomen and skin and extremities are negative, all reflexes are normal. The temporal vessels do not stand out, the radials cannot be felt, and no definite evidence of arteriosclerosis can be detected anywhere. Her urinary output is not unduly large, the chemical and microscopic examination has always been normal, the phthalein excretion is normal, and blood chemistry is normal also. Her blood-pressure varies. The systolic is 160 to 190, the diastolic 90 to 110. She is her own barometer of blood-pressure. Occipital headaches of a throbbing character tell her at once when her blood-pressure is 20 or 30 mm. higher than usual. Epistaxis has not recurred, but uterine flooding spells have been rather frequent.

She has not aged a day in ten years as far as her physical examination is concerned. Only one thing has occurred, she has become increasingly apprehensive over herself. Unfortunately her husband was afflicted with arteriosclerosis, chronic nephritis, arteriosclerotic heart disease, and hypertension for years and died shortly after she discovered her own hypertension. The mental effect of this on her can be readily surmised. For years a similar ending has hung over her head. The death of any of her acquaintances from circulatory and cardiac accidents has further served to fan the flame of apprehension in her readily receptive imagination. And unfortunately she has read and acquired a smattering knowledge of blood-pressure from news-

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florid woman who weighed 187 pounds, although her normal weight had been 205 pounds. Visible pulsation was noted in the carotids and episternal notch, her heart was enlarged, the apex being in the sixth interspace in the nipple line. Aortic dulness was slightly widened, a systolic murmur present at the base and a systolic at the apex of different intensity. Her pulse was regular, rate 104, blood-pressure 160/90. A short fat neck made palpation of her thyroid difficult and enlargement was recorded as doubtful. There was no tremor. The rest of her physical and laboratory examination was negative and she was considered a case of hypertensive heart disease with anginal symptoms. Because no definite evidence of renal or arteriosclerotic changes could be detected it was thought that her hypertension was of the essential type.

Treatment was advised and she was permitted to return to her home in the country. She returned a few days ago after three months, and you will see that she has developed other symptoms which at once indicate another diagnosis. She has not lost any more weight, but she is more nervous, looks frightened, has marked tremor of her hands, and at times you can note the distinct suggestion of exophthalmos, a characteristic staring expression and suggestion of sudden fright strike one at times. Her basal metabolic rate is only slightly elevated to +18, but the other symptoms are so striking that there remains no doubt that we are dealing with hyperthyroidism. Her blood-pressure for the past three days has been variable, one day it was 148/78, not abnormal except in the wide pulse range. Another time it was 170/90, pulse rate 122, other observations were 152/78 and 185/100. I find these fluctuations quite frequent in cases of hypertension due to hyperthyroidism and it seems to go hand in hand with the generally unstable state which these patients show. Their pulse rate, while always rapid, fluctuates sometimes within a wide range, attacks of auricular fibrillation come and go in some cases and variations in blood-pressure likewise occur. This is another disease which one should always keep in mind in connection with hypertension. I do not mean to say that all thyrotoxic cases show hypertension, but many of them do, and it should always be considered a possible cause and a careful study for other signs and symptoms of thyrotoxicosis should be made. This patient did not present a sufficiently definite group of symptoms to justify a diagnosis three months ago, but now there can be no doubt of it. She will of course have a thyroidectomy done shortly, or as soon as her condition justifies it.

**Case IV**—This next case is a young woman of forty-two who was admitted to my service recently because of headaches, nausea, vomiting, high blood-pressure, and nervousness. She has had sick headaches for fifteen years. Now sick headaches in women usually mean migraine, about which little is known etiologically. There does not seem to be any connection with hypertension. Fifteen years ago this lady's blood-pressure was found to be too high and was connected with the headaches she has had at times ever since. During the past four years her headaches have been much more severe, sometimes frontal and sometimes occipital, and her vision has gradually become affected to such an extent that, at present, newspaper print appears

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The types of hypertension illustrated by these cases are commonly seen. Many other cases might be cited but I believe these bring out most of the important points to be studied. The prognosis is often difficult, but where cardiac, vascular, and renal changes are not too advanced good judgment would seem to dictate that we do not take such a serious view of our hypertensive patients as to make nervous invalids of them in their efforts to carry out innumerable and unreasonable dietetic and living restrictions.

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In my efforts to recognize and treat this troublesome condition, I have referred freely to the literature, and what I have to say comes very largely from the experience of others, as well as my own, in the study of this single case

**Illustrative Case**—Mrs W A E aged fifty-nine years Married Native of West Tennessee. Occupation housekeeping and renting of rooms Consulted me because of following complaints loss of weight and strength, a painless diarrhea, occurring at intervals for the last four or five years, often associated with a sore tongue The attacks usually lasted for three or four days, the number of stools five or six, always occurring in the morning They were absolutely painless and, so far as the patient can tell, never contained blood or mucus The attacks lasted three or four days, and disappeared, returning in one or two weeks In the beginning there was no great loss of weight or strength, and the patient's general health was otherwise good For the past nine months, the diarrhea has become much more persistent, continuing almost constantly, until the present time During this time she has lost weight and strength, and estimates that she has lost 50 pounds She has never had any fever, but a sore tongue has been frequent During the early development of the diarrhea, the stools retained their normal color, but recently have varied from gray to light brown, with a sour, nauseating smell During the last six months there have been nausea and vomiting, occurring once or twice a week Until coming under my observation, the patient's diet consisted principally of eggs, biscuit, butter, syrup, coffee, sugar, and cream for breakfast, vegetables, macaroni, bread, butter, pie, buttermilk for dinner, and for supper she usually ate the left overs from the midday meal She has always been fond of fruit, vegetables, chicken, and fish Four months ago, in the course of a routine examination by another physician, an analysis of her gastric contents was made, and she was told that she had an absence of free hydrochloric acid Since then she has been on a strict diet, and has taken one teaspoonful of dilute hydrochloric acid in water three times a day, without any benefit The previous health of this patient has been good She never had a serious illness, except an attack of gall-stone colic thirteen years ago, necessitating the removal of the gall-bladder and stones As a result of this surgery she obtained complete relief from the colic She has had three children and one miscarriage The menopause occurred at forty years Patient has lived continuously in the region contiguous to her home in west Tennessee, except for one visit to Missouri about three years ago, and one visit to neighboring towns in Arkansas and

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Fig 196 —Photomicrograph of hanging drop preparation from pure culture monilia, forty-eight hours old ( $\times 1200$ )



Fig 197 —Composite drawing showing monilia in various phases of development—young yeast cells, single and budding, some large old cells, or dauerzellen, and two types of mycelia



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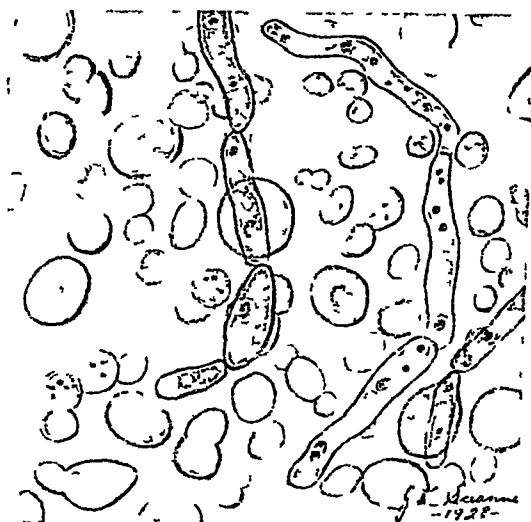


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*Direct Examination*—Hanging-drop preparations show a mixed culture consisting of a yeast and a bacillus. The yeast forms are round or oval (round forms predominate), 3 to 7  $\mu$ m in diameter, and reproduce by budding. They are bright and clear cut, appearing with a double contoured wall (Fig 196). The nucleus is distinct, and a peculiar brownian-like movement of a bacillus-like body inside the yeast is noted. In addition to the round and oval forms there are elongated forms, with slightly rounded ends which suggest

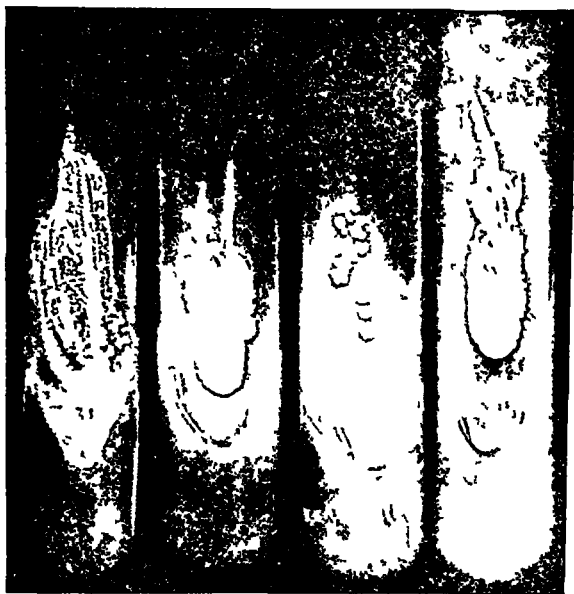


Fig 199—Six day-old cultures of monilia on Sabouraud's medium, at room temperature

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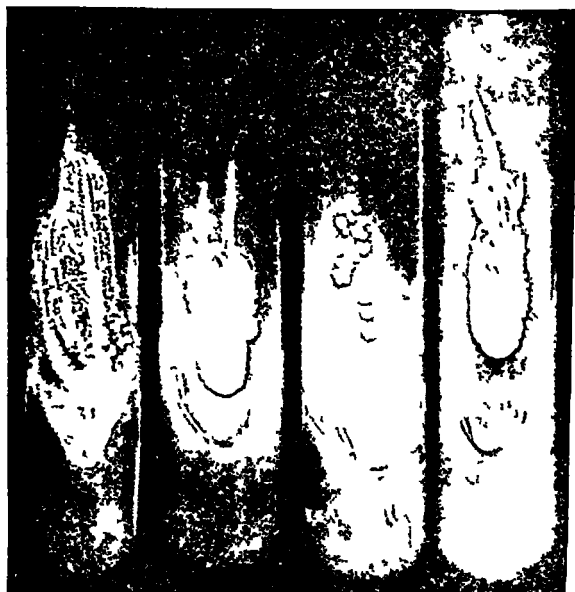


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Fig 200—Ten-day-old cultures of *monilia* on Sabouraud's medium, at room temperature

species given in the table of Castellani and Chalmers,<sup>7</sup> but the cultural and morphologic characteristics are, for the most part, suggestive of *Monilia psilosis*, and are of the subtype "C," which will conform to the work of Hannibal and Boyd,<sup>8</sup> in which types of *monilia* A, B, C have been found in sprue.

*Additional Notes*—Mycelium very poorly developed, yeast form predominates in cultures. Usual type of growth on agar slants is smooth, creamy, shining. Gelatin stab—after twenty days—does not show inverted pine tree growth, nor liquefaction. Litmus milk (reaction checked) remained alkaline—no coagulation.

Clinically, the patient presents symptoms that are strongly suggestive of tropical sprue, viz, sore tongue, secondary anemia, a painless diarrhea

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It is out of place in a contribution of this character to discuss at length the relation of yeasts to the etiology of sprue. However, it is impossible to discuss intelligently the diagnosis and treatment without a reference to some of the important facts already recognized and seemingly established by the students of the disease. Kohlbrugge,<sup>9</sup> in 1901, states that he found yeast cells in the scrapings from the tongue and esophagus. Following this Castellani, in 1905 and 1912, described several varieties of yeast found in this disease which he called *Monilia intestinalis*. This earlier work was again confirmed by Manson-Bahr,<sup>10</sup> in 1913, who found the same yeast cells in mucus from the mouth and tongue scrapings. Still later, in 1917, comes the report from Dold<sup>11</sup> in China, stating that he was able to grow yeasts in 75 per cent of normal stools, in 16 per cent of stools in patients with diarrhea, but not like that of sprue, and in 92.1 per cent of the cases that were undoubtedly sprue. This same observer was able to produce a diarrhea closely resembling sprue by feeding cultures of yeast to white mice. This last experiment of Dold's was repeated in the case of guinea-pigs by Smith,<sup>12</sup> who produced sprue symptoms by feeding a diet deficient in vitamins, and subsequently introducing *Monilia psilosis* into the animals' stomachs.

In contradistinction to the observation of the last mentioned observers, Anderson<sup>13</sup> reports that most yeasts when fed to man, pass through the alimentary canal in a living condition, not even gaining a temporary foothold. Bovaird<sup>14</sup> in this country, in personal experience with cases brought to New York City, was able to find monilia in only 1 case out of 13 under observation. His report is open to criticism, as his study was limited to direct smears from the stool, without effort to culture the organism upon suitable media.

Ashford's work has been classical, and his experience in the number of treated cases is perhaps greater than any other living author. Therefore, his conclusion that yeasts, especially the variety *Monilia psilosis*, are the chief etiologic factors in sprue, occurring in 83.6 per cent of the known cases, has the ring of authority, and must be given careful consideration, especially

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of any other etiologic factor, should be sufficient evidence for a diagnosis

The literature upon the treatment of sprue, as it occurs in the tropics, is so voluminous and varied, depending upon the authors' uncertain views as to the etiology of the condition, that it can hardly be fully discussed in anything short of a monograph. So much has been written on the subject, at such great variance, that one is almost inclined to believe there is no specific treatment, and that the disease, like amebiasis, is very difficult to cure, many patients never recovering. If all observers were in agreement that *Monilia psilosis* alone is the etiologic factor, the treatment could be attempted upon rational lines. Whether or not it is the sole etiologic factor, the fact must not be ignored that it is present in the intestinal tract in the majority of cases, and that most observers by proper cultural methods have been able to grow it in abundance from the stools. Ashford's work shows that it occurred in 83.7 per cent of the cases, and Dold's, in China, in 92 per cent.

Accepting the undisputed fact that the yeasts are abundant in the intestinal tract, the first thing that would suggest itself in the treatment would be the administration of a vaccine, made from this same yeast, or the use of an intestinal antiseptic, harmless to the patient, which would inhibit, or totally destroy, the growth of the yeast in the intestinal tract. The first measure has been tried out with varying success. A report of the work of Michel<sup>21</sup> gives much promise, but even Ashford says, "While vaccines do some good, they will not alone cure, nor prevent relapses of the disease, and they must be prepared by experts to be of any value at all." The preparation of the vaccine is difficult, requiring one well-trained in the technic, and results are so uncertain that Ashford and Michel are practically alone in their endorsement of this therapeutic measure.

Many intestinal antiseptics have been suggested, and given trials—sulphurous acid, thymol, menthol, chlorin, and quinin, calomel, perchlorid of mercury, salol, acetozone, benzosol—all with limited effect in the cure of the disease. The use of yellow santonin was suggested by Colin,<sup>40</sup> in 1878, and later on

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The literature upon the treatment of sprue, as it occurs in the tropics, is so voluminous and varied, depending upon the authors' uncertain views as to the etiology of the condition, that it can hardly be fully discussed in anything short of a monograph. So much has been written on the subject, at such great variance, that one is almost inclined to believe there is no specific treatment, and that the disease, like amebiasis, is very difficult to cure, many patients never recovering. If all observers were in agreement that *Monilia psilosis* alone is the etiologic factor, the treatment could be attempted upon rational lines. Whether or not it is the sole etiologic factor, the fact must not be ignored that it is present in the intestinal tract in the majority of cases, and that most observers by proper cultural methods have been able to grow it in abundance from the stools. Ashford's work shows that it occurred in 83.7 per cent of the cases, and Dold's, in China, in 92 per cent.

Accepting the undisputed fact that the yeasts are abundant in the intestinal tract, the first thing that would suggest itself in the treatment would be the administration of a vaccine, made from this same yeast, or the use of an intestinal antiseptic, harmless to the patient, which would inhibit, or totally destroy, the growth of the yeast in the intestinal tract. The first measure has been tried out with varying success. A report of the work of Michel<sup>21</sup> gives much promise, but even Ashford says, "While vaccines do some good, they will not alone cure, nor prevent relapses of the disease, and they must be prepared by experts to be of any value at all." The preparation of the vaccine is difficult, requiring one well-trained in the technic, and results are so uncertain that Ashford and Michel are practically alone in their endorsement of this therapeutic measure.

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In 1921 Castellani<sup>23</sup> recommended the use of sodium bicarbonate in the treatment of the disease, and suggested large doses by mouth and intravenous injections of 2 to 4 per cent solution in severe cases. The directions were to start with 1 dram three times a day by mouth, and increase gradually to 3 drams three times a day, continuing this dose for several weeks. Castellani thinks it has been of benefit to cases of sprue by decreasing the acidity of the gastric and intestinal contents, and in this way checking the growth of the monilia and other yeasts which notoriously grow so well in acid media. So far as I can tell from the literature no other observer has confirmed this work of Castellani. Others have relied upon a change in the character of the food, rather than the giving of drugs to alter the excessive acidity of the intestinal tract. Personally, I am opposed to giving large doses of sodium bicarbonate to combat hyperacidity of the gastric or intestinal tract. I believe, however, that there might be some benefit in giving other alkalis which will not produce alkalosis, *e g*, calcium carbonate. This drug might be of value in cases of sprue where the gastric contents show a hyperchlorhydria. According to the literature, the gastric contents in sprue may show a hyperchlorhydria, may be normal, may show a hypochlorhydria, or even an achlorhydria. In giving large doses of any alkali to patients whose gastric contents show an achlorhydria, harm might be done.

Pepsin and pancreatin, especially the latter, were highly recommended by the French physicians, Bertrand and Fontan.<sup>24</sup> Fens<sup>25</sup> also strenuously advocated its employment in the treatment of sprue. He claims that pancreatin acts not so much as a digestive ferment, as by stimulating the glands to whose function it corresponds. They believe that in many cases, with certain complications, these ferments may be of service, and as the indigestion is generally intestinal, pancreatin seems to be indicated rather than pepsin. T R Brown,<sup>26</sup> in 1916, reported extensive studies on the pancreatic ferments in sprue, finding all three ferments—trypsin, lipase, and diastase—absent. His technic is subject to some criticism because most of his studies were on the feces instead of the duodenal contents, as obtained through a

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ment of intestinal tuberculosis the intravenous administration of calcium chlorid was found of value. They believe that the good effect is due to the drug's inhibitory effect upon intestinal peristalsis, and its lessening of the irritability of the intestinal muscles. It is likely that calcium chlorid given intravenously, might be of benefit in the severer cases of sprue, solely for the purpose of controlling the diarrhea. The value of calcium therapy should be questioned by those familiar with the work of Ashford and Hernandez,<sup>34</sup> wherein they show that the low blood-serum calcium value in sprue is merely the result of a poor nutritional balance often occurring in other diseases besides sprue. Ashford does not believe that parathyroid extract has any effect when given by mouth, and when given hypodermically it produces only a temporary rise in the serum calcium. He believes the same rise constantly appears after dietetic treatment and vaccines, together or alone, and that there is never any need of the drug except in the rare cases complicated by tetany.

In the treatment of all types and all stages of the disease, diet is of pre-eminent importance, and is the one factor essential to success. It should be instituted early, adapted to the needs of the individual patient, and continued for months after the last active symptom has disappeared. Hence, the most important measure in the treatment of sprue is diet regulation in all cases, with or without rest in bed. All severer cases must be put to bed.

The object in dieting is to provide a plan of feeding with sufficient calories to maintain or increase the body weight, and at the same time to exclude as far as possible all starches and their derivatives, absolutely all sugar (saccharin in place of sugar when desired), and to limit fats to the minimum quantity compatible with savory cooking. The purpose of this plan of lowered carbohydrate feeding is to provide an unfavorable medium for bacterial growth, and to limit the formation of acetic, lactic, and butyric acids which Herter<sup>35</sup> has shown result from the breaking-up of carbohydrates in the intestinal tract. Fats should also be restricted on account of their laxative effect. In such intestinal conditions they are poorly digested and absorbed, hinder the

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It is interesting to note that liver, administered as liver soup, was a highly-recommended remedy, introduced in Ceylon many years ago. It can be understood now how this food may have been of value in severe cases associated with anemia, since liver has been found to be a specific in the treatment of pernicious anemia. The good effect in sprue is doubtless due to its influence upon blood regeneration, rather than upon diarrhea, and its supposed effect in increasing the flow of bile into the intestinal tract. Recently Ashford<sup>38</sup> reports upon the use of liver fraction in the treatment of the anemias of sprue, and he concludes that anemias of the pernicious type with high color index, and low red cell count (under 2,000,000) usually show definite improvement by such treatment, while those cases with anemia of the secondary type show no improvement.

Richardson and Klumpp<sup>39</sup> recently report their experience in the use of liver extract in the treatment of sprue. Their results were most favorable, for not only was the degree of the anemia lessened, but there was rapid improvement in the gastrointestinal symptoms. Basing an opinion upon their success in this case, they believe that not only can the associated anemia, but also the digestive symptoms of sprue be relieved by the use of liver extract, but they also add that it is too early to say whether or not this improvement will be permanent. This can be determined only after years of observation.

All of these diets have their good points, and in the treatment of the case presented here, a diet list was formulated composed of portions of all of the foods which had been tried by others and recommended as efficacious.

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may be increased to 12 ounces, and one or two vegetables from the following list allowed for dinner and supper

String beans	Spinach	Lima beans	{ All fresh (not canned) and well cooked Vary from day to day A serving consists of 2 to 3 tablespoons
Okra	Turnips	Tomatoes	
Cauliflower	Carrots	Egg-plant	
Asparagus	Green peas	Squash	

Raw tomatoes, lettuce, celery may be taken occasionally, with lemon juice

Avoid all potatoes, yams, red and white beans, cow peas, sugar and flour bonbons, preserved fruits, fish of all sorts, pastry, pepper, sauces thickened with flour, etc

Because of the achlorhydria in this case it was deemed best to continue the administration of dilute hydrochloric acid in doses of one teaspoonful in water after meals, in addition to the pancreatin in doses of 10 grains three times a day before meals. Under this medication, with a strict observance of the dietetic measures, the sprue symptoms have been controlled entirely. The patient is gaining weight and strength, the stools are well-formed, and rarely exceed two a day. She has returned to her home, and with a rest of two hours a day is able to carry on her duties as housekeeper.

Knowing the great proneness of sprue to show periods of improvement and then relapse, it is difficult to forecast a cure at this time. Should the patient continue to gain in weight and strength for some months, the blood approach normal, the stools continue well-formed and not exceed two a day, even if monilia are present in scant numbers, it might be considered a cure. The persistence of yeasts in the stools might place this patient in the class of a carrier. It is hoped that further study will show that the monilia have disappeared entirely from the stools, completing the cure in the strictest possible sense.\*

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There has not been entire agreement among authors who have reported cases of this disease since the appearance of Kennedy's original communication and various names have been proposed, such as "acute polyneuritis with facial diplegia," "facial diplegia in multiple neuritis," and in January, 1928 Brock and Ivimey<sup>4</sup> suggested the term "peripheral neuritis" to those cases in which only the distal parts of the nerve-trunks are affected. "Radiculitis" for involvement of the roots and "central neuritis" for involvement of cells and tracts of the neuraxis, the part to be designated by the appropriate adjective—spinal, medullary, pontile, etc.

In the matter of nomenclature in connection with the report of the case to be herewith presented, it appears to the writer that while the term "infective neuronitis" as suggested by Kennedy may have certain descriptive faults particularly in its pathologic implications, it is nevertheless far less cumbersome and sufficiently appropriate to recommend its adoption and will, therefore, be employed in this description.

Whatever name is finally agreed upon for this disease, it appears to be the general consensus of opinion that we are dealing with a group of cases in which either infective or toxic processes<sup>1</sup> are at work giving rise to pathologic changes in not only the peripheral nerves, but also the nerve-cells and tracts of the central nervous system. The disease has excited sufficient interest to have produced quite a respectable bibliography so that when Yudelson<sup>5</sup> published his contribution on the subject in February, 1926, he was able to present 67 references or articles on the disease gathered from the general literature. Since his presen-

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in swallowing although the collection of food in the paralyzed cheeks made eating somewhat of a task. There was some difficulty in speech on account of the paralysis of the lips.

The patient noticed that there was a feeling of numbness and tingling in both arms and legs. There was no disturbance of the sphincters.

Although never robust the patient had always enjoyed good health and denied the use of alcohol and occurrence of any venereal disease.

About three weeks prior to the onset of his illness patient had complained of a cold in his head and on December 29th noticed a feeling of soreness in the right calf and foot when he walked, diagnosed by himself as "muscular rheumatism." Two days later the left leg and foot were similarly affected and these discomforts have continued.

The family history was excellent except that his mother died in 1915 with tuberculosis and a sister in 1924 with the same disease.

Examination showed a tall, pale, underweight man whose face showed a mask-like appearance, with total lack of expression. He was entirely unable to walk although able to move both legs in a feeble fashion.

His tongue was moderately coated and his breath quite foul. The pharynx was somewhat redder than normal, the tonsils small and without evidence of infection. At the time of the physical examination the pulse was 100, temperature 98½° F, respirations 20.

Examination of heart, lungs, and abdominal organs revealed no evidence of disease.

On neurologic examination there was a complete paralysis of both facial nerves, with loss of taste sensation on the anterior two thirds of the tongue on both sides. Speech was not disturbed except for difficulty in pronouncing the labials. There was no evidence of dysphagia. None of the other cranial nerves gave any evidence of disturbance.

There was a very marked weakness of both legs affecting all the muscle groups, both distal and proximal, and to a lesser extent both arms in a similar fashion. In addition to the subjective sensory disturbances described it was found that there was a marked degree of blunting to touch, pain, and temperature sense in the fingers and toes and to a less extent as one proceeded up the legs and arms gradually fading into normal sensation as the trunk was approached. There was apparently complete loss of joint sensation in all fingers and toes. There was also marked tenderness on pressure along the course of the nerve trunks both in the arms and legs.

The reflexes showed definite evidence of disturbance. The knee-jerks and ankle-jerks could not be obtained nor the deep reflexes of the upper extremities. The cremasteric reflexes were not obtained, but the abdominal reflexes were present. The pupillary reflexes were entirely normal.

January 10, 1928. Urinalysis. Light amber, slightly cloudy, specific gravity 1.010, acid reaction, no sugar. There was a heavy trace of albumin. Microscopic examination showed an occasional hyaline cast. No blood present and no other abnormality.

Blood count. Red blood-cells 4,220,000. White blood cells, 11,000. Hemoglobin 88 per cent. Differential count. White blood-cells showed 30 per cent, lymphocytes 70 per cent, polymorphonuclear leukocytes.

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The patient made a good recovery with, however, some traces of facial weakness remaining after a period of nearly four months. It is not felt that the therapy in this case had any particular bearing on the recovery.

Tumors of the gasserian ganglion are of sufficient rarity to merit considerable interest and at the same time present quite characteristic a syndrome. Frazier<sup>6</sup> made a careful review of the literature to 1918 and was able to find only 43 cases recorded, of which 30 were found at autopsy and 13 at operation. Of the latter, 10 were large inoperable growths. In but 3 was the tumor of such size that it could be removed. These 3 cases were recorded by Berg, Sachs, and Frazier. In Frazier's case operation was performed about three months after the onset of symptoms. Fifteen months after operation the patient exhibited no symptoms except paresthesias in the distribution of the trigeminal nerve on the affected side.

Most of the recorded cases have been those of inoperable tumors of the middle fossa. According to Hellsten the ganglion is peculiarly resistant to invasion of tumors practically all of which take their origin not from the ganglion itself, but from the dura or some other structure of the middle fossa involving the ganglion only coincidentally. However, Frazier's operable case revealed an almond-shaped encapsulated growth overlying the ganglion and appearing to take its origin from it.

The great majority of the tumors of the ganglion are either sarcoma or endothelioma. Only one of the 43 cases was reported as fibroma.

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case the ganglion was involved in the growth of large brain tumors primarily elsewhere. Both patients suffered severe pain in the distribution of the fifth nerve, and either hyperesthesia or anesthesia over the same distribution.

A year prior to Frazier's report, Sachs<sup>7</sup> reported a case in which at operation he found a smooth tumor about the size of a large cherry, apparently completely replacing the ganglion, and the tumor at the time was considered extradural. It subsequently developed, however, that although there was for six or seven weeks relief from pain, the tumor recurred and at autopsy it was found that the ganglion had been flattened out to paper thinness under the growth which was found on histologic examination to be endothelioma. From a study of his case, Sachs felt that severe continued pain in the distribution of the trigeminal nerve with paresis of the motor branches and anesthesia over the same sensory distribution justifies at once diagnosis of tumor of the gasserian ganglion.

The following case is reported on account of the rarity of the syndrome and also on account of the extreme rarity of the type of tumor found at operation.

**Case II.**—A farmer, sixty-nine years of age, whose home was in Florida, was first seen on August 15, 1927. His chief complaints were pain in the right temple and cheek and at times swelling below and to the outer side of the right eye.

The patient was in perfect health as far as he knows until about two years ago, at which time he had an attack which was diagnosed as paralysis, producing weakness of the right side of the face and to a very slight degree the right shoulder. He also noticed that the upper eyelid on the right could not be raised voluntarily and that this condition of ptosis remained until, following medical advice, he had some type of operation performed which had the effect of lifting the eyelid.

About two or three weeks after these disturbances he began to suffer from aching pain in the right temporal region for which he was given electrical and hydrotherapeutic treatments which had the apparent effect of giving relief for several weeks. The pain, however, returned over a larger distribution, affecting now not only the region of the temple but also the right supraorbital region beginning about the hair line and gradually descending to the eye, later involving the surface of the nose on the right, the area over the right antrum, and the roof of the mouth on the right side. He also felt that the orbit itself was the seat of more or less severe pain.

The patient returned to the Sanitarium where he had been given his

case the ganglion was involved in the growth of large brain tumors primarily elsewhere Both patients suffered severe pain in the distribution of the fifth nerve, and either hyperesthesia or anesthesia over the same distribution

A year prior to Frazier's report, Sachs<sup>7</sup> reported a case in which at operation he found a smooth tumor about the size of a large cherry, apparently completely replacing the ganglion, and the tumor at the time was considered extradural It subsequently developed, however, that although there was for six or seven weeks relief from pain, the tumor recurred and at autopsy it was found that the ganglion had been flattened out to paper thinness under the growth which was found on histologic examination to be endothelioma From a study of his case, Sachs felt that severe continued pain in the distribution of the trigeminal nerve with paresis of the motor branches and anesthesia over the same sensory distribution justifies at once diagnosis of tumor of the gasserian ganglion

The following case is reported on account of the rarity of the syndrome and also on account of the extreme rarity of the type of tumor found at operation

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scaly, but there are no eruptions or gross evidences of any lesions The spine is apparently normal on physical examination

*Neurologic Examination* —Cranial nerves

I Smell seems somewhat diminished on the right and normal on the left There is, however, a slight nasal obstruction on the right which may account for the difference

II The eye-grounds show no evidence of choking of the disks or atrophy There is no evidence of disturbance of the visual fields or hemianopsia on either side

III, IV, VI The pupils are equal and regular in contour and of normal size They react to light both directly and consensually as well as on accommodation The external ocular movements are performed normally in all directions There is no nystagmus, exophthalmos, or ptosis although there is a history of ptosis on the right as well as a statement from the patient that there was diplopia three months previous, lasting one month

V Sensation of touch is definitely and markedly diminished over the distribution of the first and second divisions of the nerve Patient apparently does not feel pin prick over this distribution There is a complete loss of corneal reflex on account of a total anesthesia of the cornea

The temporal and masseter muscles cannot be felt to contract on the right side so that in movements of the jaw there is a slight deviation to the right

VII There is a slight weakness on the right occipitofrontalis muscle on the right side Otherwise there is no evidence of paralysis of the facial nerves

VIII Patient hears watch tick on the left on contact Watch tick not heard on the right There is a history of deafness on the right for forty years following typhoid fever

IX The movements of the palate are normal both right and left The gag reflex is present and there is no disturbance of swallowing

X The pulse rate at time of neurologic examination was 80, blood pressure 124/70 No history of vomiting and no evidence of disturbance of any of the branches of the vagus nerves

XI Normal

XII Normal

*Cerebrum* —There is a slight tremor of the outstretched hands but hardly more than would be expected from a feeble man of this age There have been no evidences of disturbance of memory or of any of the higher faculties

There is no paralysis of any muscle group and the strength seems good and equal on the two sides in the arms and legs

Except as noted above there are no evidences of sensory disturbances, no astereognosis or adiadochokinesia or disturbance of sensation of position of hands or feet

There is no Romberg and the gait is within normal limits There is no ataxia on any of the usual tests

*Reflexes* —The abdominal and cremasteric reflexes are equal and active on both sides The deep reflexes of both the upper and lower extremities are equal both right and left There is no ankle clonus or Babinski present There is no evidence of past or present disturbance of the sphincters

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tion the foramen spinosum was plugged with wax. The ganglion was now exposed and dissected free. A small tumor could be seen in the body of the ganglion. The sensory root was brought forward and freely dissected up from its bed. The third branch was demonstrated and divided. Then the second branch was similarly dealt with and finally the ganglion carefully dissected away from the site of the cavernous sinus, the last attachment being the ophthalmic division which was likewise divided. The whole removal was accomplished with practically no loss of blood whatsoever. The dura was now ballooned out by the injection of normal salt solution underneath it by means of a fine needle and syringe, and the wound was closed with several layers of fine interrupted silk sutures. The removed specimen showed the presence of a tumor which had the appearance of being a neurofibroma in the body of the ganglion itself.

*"Postoperative* —Patient continues to complain of some pain in the right side of the face. It is felt, however, that this is due to the fact that he has had a good deal of morphin and withdrawal of the drug has caused a subconscious desire for the drug with the production of pain. It is felt that in time all soreness and pain will disappear from the face."

A report was received from the patient on November 29, 1927 that pain in the face had largely subsided, but that at times it feels as though it was "swollen and hard." The greatest distress at this time is the discomfort in the right eye which gives him pain when exposed to bright light and appears irritated. He has gained about 12 pounds in weight, and in general health is decidedly better.

A second report was received on June 6, 1928 in which it was stated that three months previously it was necessary to have the right eye removed on account of the discomfort and evidence of irritation which was evidently due to trophic disturbances. Patient has, however, suffered no more of the original pain of the face after a lapse of nine months.

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"Sections show a very interesting structure. In the tumor area there is a well-marked cellular area, composed of medium sized hyperchromatic cells, and numerous fibers, running in various directions, with a slight tendency to form whorls. The proportion of cells to fibers is about equal, and the slow invasive character is evidenced by lack of complete demarcation. In



Fig. 204—Section through ganglion. Hyalinization, calcification, and fiber degeneration.

the ganglion itself, there is marked hyalinization with calcification and degeneration of the nerve-fibers. A few collections of lymphocytes are observed at the tumor edge. No hemorrhage or necrosis seen.

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Still other more or less distinct and less serious mechanisms of cardiac pain have, in recent years, been identified and described. Among these we consider myocardial exhaustion pain of exertion in the presence of non-dilating coronary arteries, the pain of chronic valvular lesions, of adherent pericardium, of hypertension, of paroxysmal rapid rhythm disturbances, of anemia, or methemoglobinemia, of mechanical cardiac displacement origin, and pain on a psychogenic basis with or without associated endocrine disturbances. Pain in the chest from local disease processes in the spine or thoracic wall must of course not be lost sight of. The recognition of the type and mechanism of the disorder is paramount for successful therapeutics.

### ANGINA PECTORIS

The term "angina pectoris" has been retained for the traditional dramatic type of spasmodic heart pain attack. The *classical picture* will bear detailed consideration, for entirely satisfactory clinical differentiation of the morbid state or the abnormal physiology responsible for cardiac pain is at present not always possible.

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The dramatic relief afforded these patients by the use of nitrites or alcoholic preparations along with rest is characteristic, and the attack is always aborted by the specific drugs. The precipitating factors are emotion, anger, worry or fright, eating, overdistention, flatulence, exertion, and cold drafts. Bromids in full therapeutic doses will often break a succession of attacks by dulling the sensory reception of provocative stimuli and will reduce greatly the frequency of attacks when taken regularly in 1-gram (15-grain) doses. Soporifics, as chloral hydrate in 1-gm (15-grain) doses at night or 3-gm (5-grain) doses at intervals are especially effective in quieting nervous, hypertensive individuals. Antispasmodics, as atropin in 0.5-mg (1/100-grain) doses or, better still, homatropin brommethyle in 25-mg ( $\frac{1}{2}$ -grain) doses, are often distinctly valuable in preventing and averting attacks. Vasodilators with persisting action, such as erythrol tetranitrate in 60-mg ( $\frac{3}{4}$ -grain) doses, and theocin or theophyllin ethylenediamin 0.1 to 0.2 gm ( $1\frac{1}{2}$  to 3 grains) every four hours are of value. It is only after the attempt to find out the irritative precipitating factor and removal of it is unsuccessful and the rigid application of a therapeutic régime has failed that the radical measures to be discussed later are to be considered.

#### CORONARY THROMBOSIS

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Disproportionate dyspnea, pulmonary edema with congestion and hemoptysis, cyanosis, vomiting, and hiccough are much more common in coronary thrombosis than in classical paroxysmal cardiac pain. The coronary thrombosis pain is also much more likely to be in the epigastrium and associated with other abdominal reflex states. The liver is more likely to be engorged after cardiac infarction, and further evidences of myocardial insufficiency as congestion and edema appear more rapidly. Death may come in or at a shorter or longer interval after the first breast pang.

The attack may subside, and with rest in bed the patient may recover. It is not uncommon after such a recovery to find that the patient is subject to cardiac pain on exertion. In this postcoronary thrombosis condition I have found that persistent vasodilators, such as theocin or theophyllin ethylenediamin increasing the coronary circulation, aid greatly in the reparative processes and increase the exercise tolerance and preclude the attack. The drugs should be given in 0.1 to 0.2 gm ( $1\frac{1}{2}$ -3 grains) doses every three or four hours. Complete solution of the preparation is a most essential detail and is the deciding factor in the success or failure of the effectiveness of the drug.

#### MYOCARDIAL EXHAUSTION PAIN

*Sclerosed coronary arteries* apparently cannot and do not respond with dilatation after exertion. The usual threefold coronary circulation required by the heart after exercise is not forthcoming and myocardial anoxemia and stretching fatigue result. Pain of a somewhat less sharp character, due to the so-called ischemia cordis intermittens, and accompanying distention of dilatation develop. Distress of this source is promptly relieved by rest. Vasodilators, the action of which persists, especially theocin or theophyllin ethylenediamin are of value in this condition.

*Certain advanced chronic valvular diseases, especially mitral and*

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In spite of the fact that the patient has often learned that the taking of sodium bicarbonate facilitates the eructation of the gas from the stomach and relieves his distress, yet he worries until he is reassured and convinced of the origin and mechanism of the disturbance. The patient who suffers from this condition is usually of the hyperesthenic habitus with a transverse heart due to a high, flat diaphragm, a protuberant abdomen, and an increased intra-abdominal pressure. The symptoms are apparently produced in the pushing up of the dome of the left leaf of the diaphragm by the distended fundus of the stomach. The already transversely placed heart resting on the dome of the diaphragm may be shifted posteriorly. In this shifting a slight twisting of the heart, sufficient to embarrass slightly the coronary circulation, may take place, and give rise to symptoms, as will be explained later.

The predisposing habitus position of the heart, evidence of its shifting, the presence of abnormal gastric tympany in the right hypochondrium, extending Traube's semilunar area considerably, favor the diagnosis of a mechanical or postural cardiac embarrassment. The relief by change of position and belching, along with the absence of any discoverable cardiac or vascular abnormality, must be considered sufficient basis for the diagnosis.

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A *neurogenic form of more or less benign precordial distress* is described by individuals, usually females of a high-strung, *neurotic temperament*. The abnormal sensation varies from a heavy subcardiac ache to sharp, sudden, instantaneous precordial pains that momentarily transfix the thorax or radiate to the left axilla or left shoulder and sometimes even into the left arm. Ectopics or premature contractions of the heart are usually noted, and will account for the sudden, sharp, momentary pains in these very sensitive individuals. The patient is usually erethistic, restless, and emotional. Irregularities, tachycardia, palpitation, throbbing sensations, giddiness, and even vertigo are common symptoms which are, however, rarely present in one critically ill with the serious heart pain. The attacks are usually relieved by exertion and the patient has a good exercise tolerance. Except for the rhythm disturbance, the cardiovascular system is entirely negative in this condition. It is unfortunately also true that some cases of true anginal syndrome present very little abnormality on physical examination.

#### THE MECHANISM OF PAIN

The *classical syndrome of angina pectoris*, as described, is the condition that warrants great respect as well as careful study for clear understanding and accurate differentiation. The symptom complex presents phenomena that are in a large measure neurologic. The explanations of the mechanism of the production of the cardiac pain is still for the most part theoretical.

Among the theories to account for cardiac pain, the vascular spasm idea with proximal stretching of the coronary artery and distal ischemia must still be considered tenable. Circular constrictions with proximal dilatation and distal blanching are to be seen in the retinal arteries in individuals subject to cerebral episodes. This is convincing evidence to me that such vascular phenomena do occur. Whether the pain is the result of the stimulation through the sensory nerves in the adventitia of the coronary artery or of the stretching of the ischemic ventricular

arrhythmia with or without ectopics or premature contractions, likewise promptly disappear after stopping the use of tobacco

A *neurogenic form of more or less benign precordial distress* is described by individuals, usually females of a high-strung, *neurotic temperament*. The abnormal sensation varies from a heavy subcardiac ache to sharp, sudden, instantaneous precordial pains that momentarily transfix the thorax or radiate to the left axilla or left shoulder and sometimes even into the left arm. Ectopics or premature contractions of the heart are usually noted, and will account for the sudden, sharp, momentary pains in these very sensitive individuals. The patient is usually erethistic, restless, and emotional. Irregularities, tachycardia, palpitation, throbbing sensations, giddiness, and even vertigo are common symptoms which are, however, rarely present in one critically ill with the serious heart pain. The attacks are usually relieved by exertion and the patient has a good exercise tolerance. Except for the rhythm disturbance, the cardiovascular system is entirely negative in this condition. It is unfortunately also true that some cases of true anginal syndrome present very little abnormality on physical examination.

#### THE MECHANISM OF PAIN

The *classical syndrome of angina pectoris*, as described, is the condition that warrants great respect as well as careful study for clear understanding and accurate differentiation. The symptom complex presents phenomena that are in a large measure neurologic. The explanations of the mechanism of the production of the cardiac pain is still for the most part theoretical.

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There is still considerable difference of opinion and some confusion as to the details of the paths

The coronary and the aortic deep and superficial plexuses are derived from sympathetic fibers, the so-called inferior, middle, and superior cardiothoracic nerves from the ganglia of the upper thoracic sympathetic cord. Afferent fibers to the central nervous system are apparently carried to the brain by way of the vagus and the depressor nerve, which is apparently intimately associated with it in the human being. Impulses go to the spinal cord through the white ram of the upper three or four thoracic, the stellate, the inferior, and the middle cervical ganglia. Vasomotor fibers to the coronary arteries have been shown to accompany the cardiothoracic nerves.

The radiation of the pain is easily explained on the segmental distribution of the different cardiac fibers. The usual spread is felt in the nerves from the upper four dorsal segments, but sometimes as low as the sixth dorsal and the seventh and eighth cervicals with pain in the neck and the back of the head. The first and third dorsals go to the retromanubrial region, the ulnar aspect of the forearm and arm, while the second dorsal segment is distributed to the inner aspect of the arm to the elbows.

#### THE SURGICAL TREATMENT OF HEART PAIN SOME INDICATIONS AND CONTRAINDICATIONS

Obviously, there are refractory cases of heart pain which do not respond to medical treatment. In the hands of a skilful therapist the unsuccessfully treated cases are relatively few in number. Among these unrelieved patients certain ones may be considerably benefited by a surgical blocking or severing of the nervous pathways.

The cases most favorable for surgical treatment from the medical standpoint are, of course, those with frequently recurring refractory paroxysmal pain of an excruciating nature, but without or with little evidence of organic disease in the aortic root, in the coronaries, or in the heart-muscle. The spasmodic type is the most amenable to neurosurgical treatment. Only where a dramatic nervous mechanism precipitates the

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sometimes does, the surgical method should be resorted to, for the ultimate aim is to stop the painful crises. The attacks of pain are far from innocuous for the patient may die in any attack as a result of myocardial ischemia, anoxemia, and its fatal complication, ventricular fibrillation. This can occur without coronary thrombosis, and sometimes with little or no gross change in the coronary arteries or the heart. There lies in the crises of pain far more danger, I feel, than could possibly come from the removal of the pain which has been characterized as the "danger signal, the cry of anguish of an impoverished myocardium." Limitations must, of course, be indelibly impressed upon one who has been relieved of pain. It must be remembered that in the removal of sensory fibers, motor vasoconstrictors are also cut and the dangers of critical crises are less, but the disease process remains. It has even been suggested that repeated, painful crises may serve to exaggerate and hasten the basic pathologic processes.

The ideal case of heart pain for surgical intervention is usually the type that is a promising prospect for medical treatment, and as a rule these patients will not consider surgical treatment until medical therapy has proved useless. The surgeons prefer as subjects patients who have infrequent attacks, and who present no evidence of organic changes in the aortic root, the coronaries, or the heart valves.

In such patients it is true that the operative mortality will be kept low, and the therapeutic method will be less likely to fall into disrepute if the selection of cases be most rigid. If only the patients with the shorter attacks of pain at longer intervals and only on exertion of a considerable degree with no evidences of myocardial insufficiency between attacks and no electrocardiographic abnormalities be submitted to operation, the odds would be distinctly favorable for this method, or any other method of treatment that might be applied. Indeed if these restrictions are adhered to the number of surgically treated cases will of necessity be small and unrepresentative, for then there will undoubtedly be a preponderance of purely neurogenic cases. As a matter of fact, if the surgical treatment must be restricted to

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paroxysmal ventricular tachycardia, and high-grade fibrillation should make one hesitate and weigh carefully all the facts of the whole situation before advising surgical intervention for the relief of pain. To be sure, patients presenting any of these abnormalities are less safe surgical risks while a patient without them is a good risk. Selection on this rigid basis will keep the immediate operative mortality at an insignificant level, but it will also, I fear, deny the boon of relief to sufferers for many of whom life has become a painful burden. To these pain-tortured souls, exitus is looked forward to and existence is so miserable and agonizing that anything is welcomed.

**The Surgical Operation**—The choice of the operative procedure must be guided by the condition of the patient. In the patient that measures up to the ideal requirements as a surgical risk, Cutler's statistics seem to indicate that the extensive ablations of the entire cervical sympathetic chain, including the first thoracic ganglion at once interrupting sensory and vasoconstrictor fibers, first on the left then on the right, offers the greatest hope of lasting relief. Relief was afforded even in the advanced severe type of the disorder. The complete unilateral procedure was well borne even by the patients with the serious type of the disturbance. In the most severe cases Cutler considers it wise to perform a partial sympathectomy, the removal of the superior cervical ganglion, division of the superior cardiac nerve, and that of the ramus communicans as a first stage to a subsequent complete sympathectomy. These two procedures have been most popular in this country. Partial cervical sympathectomy may be limited to the middle or inferior ganglion or to the stellate ganglion. Leriche has advocated the division of only the rami communicantes of the stellate ganglion through which pass the majority of the cardiosensory fibers to the spinal cord. The trunk directly above the stellate ganglion is usually also cut along with interruption of the vertebral nerve and the depressor fiber when it can be identified. The Viennese group advocated the section of the depressor nerve only on the basis that it is the chief afferent sensory nerve. The irregularity of the presence of this as a distinct cord, the difficulty of its identification, and the ques-

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The cases are of further interest in that both were females, and females with heart pain are much more uncommon than are males with the same trouble. Case I dated her trouble from an operation for gall-stones, which is unusual, while the second patient was seized while at hard work and apparently suffered a heart strain. The first patient presented a status anginosus and definite evidences of congestive heart-failure, while Case II had beginning congestive failure. Both had hypertension and enlarged hearts, aortic systolic murmurs, and abnormally metallic aortic second sounds, strongly suggesting aortitis at the root of the aorta, with the sclerotic processes more than likely involving the coronary arteries especially at their orifices. Abnormal electrocardiographic findings with persistent negative T waves were evidences of myocardial damage in both cases.

The second patient who was apparently the better risk and who was subjected to the least trauma and ablation, suffered the more stormy course after the operation, and had the more serious complication.

The postoperative courses were further evidence of the presence of advanced coronary disease most probably with healing myocardial infarction from the previous thromboses. In Case I there seemed to be little doubt of a thrombosis of the coronary having occurred. In Case II it is difficult to decide, however, whether or not coronary thrombosis had taken place, yet the stormy postoperative course with possible embolism was considered proof enough that such had been the case. The pain in each instance may be considered of the postcardiac infarction type, the first episode in each instance having been precipitated by a coronary thrombosis. The association of a glycosuria in the severe paroxysms of cardiac pain in the first case is indeed interesting and should have been more completely studied.

**Case I Angina Pectoris, Probably Coronary Thrombosis with Status Anginosus, Greatly Relieved of Pain by Partial Sympathectomy**—R. S., a married Russian Jewess aged forty five years, came to my out patient clinic because of pain in the heart of a constant, boring type with occasional sharp exacerbations, shortness of breath, and swelling of the dependent parts. She

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The heart area of dullness was apparently considerably increased extending almost to the anterior axillary line The apex impulse could not be seen or felt An aortic systolic murmur, a loudly accentuated aortic second sound, a slight tachycardia, and numerous premature contractions were the only abnormalities that were detected The brachial and radial arteries showed moderate sclerosis The blood-pressure was greatly elevated and rose some during the attacks

The *electrocardiograms*, taken in the service of Professor Frank N Wilson to which the patient was transferred, showed slight left ventricular preponderance, ventricular premature contractions, inversion of T waves at first in lead I and then in I and II, and finally in all three leads with evidences of intraventricular block

The abdomen was extremely obese with an apron like fold over the symphysis The scar of what had been a low gall-bladder incision was present The extremities showed a slight subcutaneous edema

The laboratory examinations were of no significance except for the finding of a heavy glycosuria during the crises of pain, but none between attacks The non-protein nitrogen was normal, 33.7 mg per 100 cc of blood The Wassermann reaction was negative

*Progress Notes*—Morphin was required in addition to amyl nitrite and nitroglycerin for relief from the attacks The patient was transferred to the surgical service for sympathectomy Dr F A Collier removed the *superior* and *middle cervical sympathetic ganglia* with the intervening cord A mass of adjacent inflammatory cervical lymph-nodes made the operation difficult After the operation Horner's syndrome (slight ptosis of the left eyelid, and pupillary dilatation) were present The ganglia, on microscopic study, were found to show no degeneration and no abnormality except for a small collection of lymphocytes

*Discussion of Case I*—As a result of the operation the attacks of pain had lost their knife-like severity, and had changed considerably in character Her pulse was down to 80 with the symptoms definitely relieved one month after operation Three months after operation the patient returned to me because of attacks of heavy palpitation, choking sensation, and dyspnea, but with no return of the sharp pains She was frequently aware of the dull precordial sensation of an aborted attack which before the operation was always associated with excruciating pain She died in an attack three years after the operation

**Case II. Angina Pectoris, Possibly Coronary Thrombosis, Relieved by Partial Sympathectomy, but Complicated by Postoperative Gangrene**—W E, an American housewife, aged fifty-seven years had been seen by Professor Frank N Wilson in consultation and referred to the hospital for

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The abdominal panniculus was very thick. The liver and spleen could not be felt, and no tenderness was elicited below the costal margin. The reflexes were all normal in the extremities. There was no edema present.

The Wassermann reaction was found to be one plus on one occasion and was not repeated. The other laboratory studies were negative. The x-ray films showed pleural thickening at the right base. There was no fever present at any time while she was in the hospital.

*Progress Notes*—During the first few days of hospitalization slight exertion in bed precipitated severe anginal attacks on several occasions. Nitroglycerin under the tongue in 0.5-mg (1/100 grain) doses relieved the attack. Barbitol in 0.6-gm (10 grain) doses seemed to ward off night attacks.

The patient was transferred to surgery and operated upon by Dr. F. A. Coller. The middle cervical sympathetic ganglion was quickly exposed and skilfully removed. There was a minimum of trauma and the operation took very little time and seemed to be a simple, innocuous procedure. After the operation there was some pain in the surgical wound, but no return of the paroxysmal attacks of pain. It was observed by Dr. Frank N. Wilson that the hyperesthesia over the precordium was greatly decreased.

Three days after operation she was suddenly seized with a severe, sharp pain in the left foot and leg. The left foot became gangrenous. The complication was considered to be the result of an embolus from a mural thrombus in the left ventricle. Amputation was done through the lower third of the left thigh. The patient later developed erysipelas of the face, but recovered from this as well as the amputation. During the two months after operation she suffered no further anginal attacks.

Dr. C. V. Weller reported that the histologic study showed much fibrosis of the nerve sheaths, but he found no ganglion cells in the level sectioned in the pathologic laboratory. The left foot showed early anemic gangrene without infection. There was a thrombus in a large vein and evidence of congestion in others. The vessel walls were moderately thickened. The large peripheral nerve showed slight inflammatory infiltration.

*Discussion of Case II*—The painful cardiac seizures were apparently stopped and did not recur in the two month period of observation. The complications were entirely unexpected, and this patient was considered a better operative risk than the first one, and less surgical ablation was done since only the middle cervical ganglion was extirpated. This was apparently enough, however, to give relief. The hospitalization must, of course, be given its share in any therapeutic evaluation. This is, however, a difficult factor to estimate. The amputation of the left leg was well borne, and this served further to restrict the patient's activities. An embolic hemiplegia often serves a similar purpose and hampers and restricts a patient who otherwise might not

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Out of the maze of information that is being brought forth under this new stimulus of surgical sympathectomy of various types for the relief of pain should come some clear conception of the pathways of cardiac pain. The clue to the mechanism and etiologic factors concerned in the classical, apparently neurogenic angina pectoris may eventually be forthcoming. The method warrants our careful attention and study. To some of our patients it offers a great boon.

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The obvious in this case is that we have a patient with a constitutionally inferior psychic makeup, that she has been exposed to failure, disappointment and domestic difficulty, that she has driven herself to efforts beyond her strength, and that subsequently she has developed visceral neuroses of unusual intensity. The problem that interested me most, however, was why the neuroses took the form of spasm. Perhaps this question would never have suggested itself to me, but for my stay in China and my observation of the common ailments of the Chinese. Among the Chinese the everyday clinical picture that I have just sketched is not seen. I didn't see it. Excellent clinical observers who have been long in China tell me they did not see it. On reaching Peking I was told by Dr. Robertson who held the Chair of Medicine in The Peking Union Medical School that a case of angina pectoris had never been seen in a Chinese.

In China as elsewhere, all the types of heart disease are seen. Every day one can see rheumatic hearts, syphilitic aortitis, hyperthyroid hearts, fibrillators, heart lesions associated with the contracted kidney. The question is worth asking—does the fact that the Chinese are not subject to angina pectoris lighten the difficulty of comprehending the anginas we see here?

A collection has been made of as many as eighty different theories to explain the syndrome of angina pectoris. Those with which we are most familiar are that of Mackenzie, which, leaning on the conceptions of Head, would indicate that angina pectoris is not properly speaking a heart pain, but the expression of a series of viscerospinal reflexes, and which attributes the clamorous response of the cord to the violent excitation of the centripetal fibers—that of Allbutt who thinks that distention of the aorta is the key to the problem, the theory most generally taught—that of coronary thrombosis, to which the chief objection as a

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would lead us to expect what actually develops, that the author is bound to no procrustean bed of theory in his exposition of the essential nature of the anginas. "The whole history of angina pectoris," he remarks elsewhere "from the time of the first observations down to the assemblage of volumes that have appeared in recent years is marked by the same evil—to wit, the vain pursuit of the morbid entity that lies beneath the fugitive phenomena of the syndrome." It is from him, however, that I have taken the words "spasmogenic aptitude" that form the title of my remarks. A certain spasmogenic aptitude must be assumed to explain the incidence of anginal pain, the underlying pathology in any case being grave, slight, or none at all.

If the spasmogenic aptitude is great, a slight stimulus of neurogenic nature will suffice to evoke the spasm with its characteristic pain. If the spasmogenic aptitude is absent, the utmost stimulus will fail to evoke the spasm. Between these extremes, stimuli will provoke spasm in accordance with the ratio that exists between spasmogenic aptitude and the vigor of the stimulus.

The term "spasmogenic aptitude" would seem to have about the same meaning as spasmophilia, but spasmophilia has attached to it certain connotations relating to calcium metabolism and the activity of the parathyroids. We would like to say that the Chinese do not have angina pectoris because they lack the spasmogenic aptitude without saying that their parathyroids are inactive. We would like to say that the majority of our cardiopaths do not have angina pectoris and that the minority that suffers from angina suffer by reason of this spasmogenic aptitude without meaning that they are deficient in calcium.

To recur to the patient whom we are considering we will recall that she not only had anginal pain, but several attacks of abdominal pain that kept her busy all night with enemas and stupes working for relief. If a person has a constitutional aptitude toward spasm in unstriated muscles it might be expected that unstriated muscles in different systems would be affected. This expectation is quite generally realized. When I look back over the patients I have seen suffering from the benign anginas

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**Case II. Spasm of Gullet**—B X, aged thirty-seven years, 6 feet, 2 inches in height, weight 176, of rugged muscular build. Manages a tailoring shop. Was treated by me twelve years ago, when he was a farmer, for severe discomfort after eating and fear of taking food. At that time he weighed only 118 pounds. He made a prompt and good recovery. Being ambitious he pressed ahead in the world until he had now come to have a good business.

He entered my consulting room with his collar unbuttoned and his necktie knotted loosely far below its proper place. This he said was due to choking. Anything touching his throat made him choke. Shaving was most distressing to him and he could by no means lie in a barber's chair. What distressed him most was riding in a car. As soon as he put his foot on the clutch his throat clutched. In busy traffic his plight was worse.

The trouble had come on suddenly about a year previously. While riding at fifty miles an hour toward his country home he felt his throat close up. He stopped the car and pulled out to the side of the road. His heart was beating tumultuously. Ever since his life had been miserable. His sleep became broken. He lost 15 pounds in weight. His discomfort after eating returned. He recalled the maxims and measures that had helped him before but they failed him.

He was physically sound every way and treatment by moral persuasion and encouragement, an ordered life, and mild sedatives soon restored him to comfort. This seems to have been an intensified form of globus—perhaps the commonest of all the spasms.

**Case III. Spasm of the Cardia**—C D, a vigorous man of forty eight, a county policeman, gave a history of having had three attacks of inability to swallow food or any water. Attempts at swallowing were followed by immediate regurgitation. The attacks had lasted each time nearly two days. In his last attack he started to the city, a sixty mile drive, to see me. When he entered he still thought he could not swallow. A semisolid barium meal and a stomach-tube passed through to his stomach without any evidence of obstruction. The attack—apparently one of spasm of the cardia—has passed away. This type of spasm, unlike most, causes frequently no pain, merely offering an obstacle to taking food. General physical examination—no important findings.

**Case IV. Spasm of the Midesophagus**—J M, a brisk business man of thirty-four, alert in bearing, lively in speech, of slender but adequate build. He comes with a ready made diagnosis of peptic ulcer. Examination shows moderate hypermotility and hyperacidity of the stomach, but no filling defects and a good cap. His complaints, it turns out, are not of the stomach, but of substernal pain—a sudden pain as of a huge ball forming in the breast, swelling as if to burst, accompanied by a tightening in the throat and a feeling of impending disaster. He must stop still till the pain subsides. The pain is not related to taking food. It may come when he is drinking water, or again without any provocation when he is walking along the street.

This kind of pain, while it is not easy to prove the fact, objectively is almost surely a spasm of the esophagus. A hot drink tends to relieve it. Rest and relaxing treatment cure it fairly easily.

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Though she is of a lively temperament and socially active, and despite a not wholly well adjusted home environment, she was fairly well after her operation until she contracted amebic dysentery. This was accompanied with severe pain in the left colon and a very vigorous treatment with emetin and stovarsol contributed to reduce her nervous potential. She finally became free of all evidences of dysentery except spasmodic pain which tormented her cruelly at the most unexpected times. When seen four months ago superficial ulceration was found in the upper rectum and lower sigmoid. The junction of descending and pelvic colon was very sensitive to pressure.

Despite rest and sedatives, together with careful attention to diet and evacuation of the colon, the spasm occurred at intervals of ten days or two weeks with the greatest violence. The best relief from the excruciating pain was found in large doses of chloral, which seems to be our most reliable anti-spasmodic. A more recent repetition of the x-ray and sigmoidoscopic examinations shows the mucous membrane now healthy, the haustra of the colon well defined. Two barium enemas showed the colon capable of good dilatation at the point of maximal tenderness. The pathology seems to be very slight but the spasms have not yet been overcome.

There is something in the way in which the spasm recurred suddenly after a period of relative well being that made one think of epileptic seizures, the more so as this disease had been manifested in the family history.

**Case VI Spasm of the Bladder Mechanism**—A young man of twenty six years, a gentleman farmer, entered complaining of spasms in the colon. These were evidently immediately due to misuse of cathartics. Five months previously he had been operated on for perforated duodenal ulcer from which he made a good recovery so that he had evidently had spasm in the pyloric region. His father had also had a long siege with pylorospasm and gastric ulcer with hemorrhage, which finally required surgical treatment. This young man next appeared with a spasmodic stricture. He had no history of, and no evidence of, venereal disease. The spasms in the cut-off muscle were somewhat painful, but the chief difficulty was interference with micturition. In the course of time with forced feeding and rest he became free from all his spasms, though the usual treatment with sounds helped the spasm in the urinary tract.

**Case VII**—A Jewish merchant, aged thirty years, bachelor, in sound general health. Complained of urinary frequency whenever he was in company, though not at other times. He had gradually shut himself off from theaters and all social gatherings. He never called at the homes of his friends on account of this embarrassing symptom. It was obvious in this case that the troubles were purely psychogenic.

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Essential hypertension is unknown in China. Cases of hypertension though uncommon there are found associated with the contracted kidney but the arteriosclerotic kidney does not seem to be caused by prolonged hypertension.

I saw in China one interesting exception to this general rule. A supervisor of nurses aged thirty-five who had been trained many years in America and had acquired the American quickness and alertness of movement, in striking contrast to the slow, easy ways of the average Chinese nurse developed an essential hypertension and severe headaches. She had so alienated herself from the native Chinese attitude that she used to refer to "these Chinese" with their trifling ways. Her thorough-going and energetic demeanor had brought her into an essential hypertension. In a large service extending over four years this was the only case I ever saw of essential hypertension in a Chinese.

It is well known that the Chinese normal blood-pressure is 100/65, and observations have been made to show that when they live in America they acquire our normal pressure. Dr J H Foster of the Medical College of Yale-in-China who studied the blood-pressure of a group of foreigners living in China for a number of years, found that their blood-pressure generally in the course of time came down to the Chinese level. Other observers have noted the same phenomenon. When a foreigner living in the interior of China acquires an essential hypertension it is generally obvious that the sufferer is a person living in a state of antagonism with his environment—one who has come to China to teach everything and learn nothing.

There is a question whether a continuous and tonic spasm such as we see in essential hypertension should be classified with the briefer spasm that we have been considering. We may remember, however, that the digestive tract also has its prolonged tonic spasm as seen for example in cardiospasm and much more commonly in the tonic spasm of spastic constipation.

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### DISCUSSION

We have cited briefly a number of cases showing an aptitude to spasm in the unstriated muscle, in the circular coats of the various conducting tubes in the alimentary tract, the urinary tract, the blood-vessels. We have called attention to the very great frequency of spasm in our people as contrasted with the relative infrequency of this symptom among the Chinese.

What can we say as to the significance of this observation? It is quite certain that in some instances the spasm could be directly referred to psychic states—were directly traceable to low nerve potential—to chronic fatigue, others to disturbances in the emotional life, or to the force of suggestion. There are cases in which there is present, it is true, a minimal degree of morbid pathology, but in which the pathology is quite inadequate to explain the violence of the spasmodic reaction, cases which contrast strangely with others having an extensive pathology of the same type, yet without spasm.

It may be taken as an accepted dictum of physiology that the circular coats of these tubes, the contraction of which produce the painful spasms referred to, whatever inherent quality of contractility they may possess contract in the main as the result of stimulus coming through the vegetative nervous system. The vegetative nervous system is the proximal cause of the trouble. At this point we inevitably begin to think of the theories of Eppinger and Hess. Is it vagotonia or sympathicotonia that we have been discussing all along? Without attempting to rehearse the arguments pro and con, it will be enough to say that the analysis of this question by Cannon, Langdon Brown, and others suffices to make it clear that no such schematic division as Eppinger and Hess have undertaken can be maintained. We often in the same person, at the same time, meet with spasms that belong to the sympathetic and parasympathetic overaction.

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believe, that while the poor take much less protein than we do and that vegetable protein, the well to do seem to eat as much meat as we do. Their cooking is elaborate and epicurean, ours by comparison is simple and unsophisticated, but the proportions of the essential components of diet among the more affluent Chinese is about the same as with us. As between the coolie in China, living on his rice and herbs, and the wealthy gentleman sitting down daily to a highly-seasoned menu of fish, fowl, mutton, pork, and beef no differences were observed in blood-pressure or other tendency to spasm. Further, the considerable group of occidentals living in the interior of China, whose blood-pressure sank to the Chinese level took practically the same diet as at home. There are seen in China many morbid conditions related to diet and metabolism—beriberi, rickets, xerophthalmia, on the one hand, diabetes and obesity on the other, but the spasmogenic aptitude is lacking.

It is attractive to muse on the possibility of some endocrine imbalance as representing the kind of chemical stimulus that acts on the vegetative nerves to provoke spasm. Are Chinese lacking in adrenal or in pituitary? Have the sufferers from visceral spasm an over supply of incretion from these glands? One thinks of the hairlessness of the Chinese breast and limbs, of his irenic disposition, but then one reflects on the absence of any constant marking of the spasmogenics with evidence of glandular excess and this line of conjecture loses itself.

A far more helpful lead in our search for a way of thinking about the essential nature of the spasmogenic aptitude is offered by the promise of psychologic analysis to reveal the *primum movens*. Ah yes, the Chinese lack the spasmogenic aptitude—placid, gentle, peaceloving—Buddhist—their ideal, the serene calm of Amida Buddha, with closed lids and folded hands—symbol, the lotus flower scarcely swaying over the still pool. We are so well acquainted with the high-bred Chinese in fiction, a monstrous mixture of atrocity, suppleness and suavity with his jade ornaments, his silken embroideries, his naïve disarming smile, behind which diabolism lurks—so assured and self contained that his pulse never quickens as he plans and executes

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We seem to have fallen on contradictions—just what is calm? The first association is with surface—calm sea, calm lake, calm visage—“calm yourself”—that is, reinforce your inhibitions and acquire external immobility We have all seen essential hypertensives who outwardly were calmness itself It must be that there is an inner calm, a deep calm, possible even in conjunction with superficial agitation

A Chinese expressed the fundamental distinction between Chinese and occidental psychology by saying that the occidental when meeting with a difficulty tries to remove it, making a change in the external environment, an external adjustment—the Chinese finds it easier to make an internal adjustment—to change his soul to an attitude of acceptance When the axle on the wheelbarrow squeaks an squeals we grease it The Chinese wheelbarrows are never greased They can be heard half a mile away We say the screeching is intolerable—they accept it If we stay in China long enough we, most of us, learn to accept things, and our normal systolic pressure becomes, as the Chinese is, about 100 If the Chinese live in America long enough they learn our habit of protest and non-acceptance, and their normal blood-pressure rises to our level

The suggestion naturally arises—if we would avoid spasm be as the Chinese True enough, but all our ideals of progress and development are bound up with the zestful spirit of protest and non-acceptance We must realize that a low reaction threshold to spasm is not an unmixed good The spasms from which we suffer are the “defects of our qualities,” a precious racial asset become excessive and gone awry

What to think of the essence of the spasmogenic aptitude We have found ourselves forced to place it in the dim category of constitutional defects—constitution made up, as it is, of something inborn, something taken from environment

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tongue appeared entirely normal. The mucous membrane of the entire cavity was pale, there was no pigmentation. The head presented nothing else of note. The neck, the thoracic wall, and the lungs were negative. The heart was not enlarged, there was a systolic murmur, which was not constant and which was not transmitted, accompanying the first sound, the second sounds were clear and not accentuated. The blood-pressure was 122/90. The abdomen was very much distended, ovoid in shape, and tense to the palpating hand, no masses, tumor, or organs could be felt, and there was no tenderness or rigidity. The classical physical signs of free fluid in the peritoneum were easily elicited. The genitals were negative. The lower extremities were edematous to the knees. The patella reflex could not be elicited. There was no local or general adenopathy.

*Clinical Course*—The patient's general condition improved rapidly and steadily. The edema disappeared in about ten days. The improvement in the blood-picture was very prompt, as shown by the blood reports, and was typical of the response to be expected from liver feeding in pernicious anemia. There has been no fever and little change in pulse rate or blood-pressure. The ascites has shown no improvement. Two gallons of a clear straw fluid was removed by paracentesis November 12, 1928, the fluid rapidly reaccumulated and 4500 c c was removed November 24, 1928 and a like amount on December 12, 1928. At the present time, December 22, 1928, the abdomen is again much enlarged. No therapy other than liver feeding and rest was instituted. The use of novarsurol was considered but not instituted. The fluid output was much in excess of the intake during the early part of the patient's treatment.

*Laboratory Examinations*—Blood Examinations

11/ 2/28 Total red cells per cubic millimeter, 990,000 Hemoglobin, 40 per cent

Total white cells per cubic millimeter 6250 Color index, 2.2

Differential count S M 50, L M 0, E 0, B 0, N 50

11/ 6/28 \* Total red cells per cubic millimeter 1,420,000 Hemoglobin 41 per cent or 5.82 gm

Hematocrit volume 18.5 per cent

Average corpuscular volume 130.3 cubic microns

Average corpuscular hemoglobin 41 x 10

Percentage of hemoglobin in average cell 31.5 per cent

Volume index 1.59 Color index 1.43 Saturation index 0.9

Price Jones count average of 130 cells, 8.7 microns

Numerous megaloblasts, some normoblasts, reticulocytes

Anisocytosis and poikilocytosis very marked

11/16/28\* Total red cells per cubic millimeter 2,340,000 Hemoglobin 56 per cent or 7.96 gm

Hematocrit volume 28.8 per cent

Average corpuscular volume 123 cubic microns

Average corpuscular hemoglobin 34 x 10

Percentage of hemoglobin in average cell 27.6 per cent

Volume index 1.50 Color index 1.19 Saturation index 0.79

\* These examinations were made by Dr. Max Wintrobe

tongue appeared entirely normal. The mucous membrane of the entire cavity was pale, there was no pigmentation. The head presented nothing else of note. The neck, the thoracic wall, and the lungs were negative. The heart was not enlarged, there was a systolic murmur, which was not constant and which was not transmitted, accompanying the first sound, the second sounds were clear and not accentuated. The blood-pressure was 122/90. The abdomen was very much distended, ovoid in shape, and tense to the palpating hand, no masses, tumor, or organs could be felt, and there was no tenderness or rigidity. The classical physical signs of free fluid in the peritoneum were easily elicited. The genitals were negative. The lower extremities were edematous to the knees. The patella reflex could not be elicited. There was no local or general adenopathy.

*Clinical Course*—The patient's general condition improved rapidly and steadily. The edema disappeared in about ten days. The improvement in the blood-picture was very prompt, as shown by the blood reports, and was typical of the response to be expected from liver feeding in pernicious anemia. There has been no fever and little change in pulse rate or blood-pressure. The ascites has shown no improvement. Two gallons of a clear straw fluid was removed by paracentesis November 12, 1928, the fluid rapidly reaccumulated and 4500 c c was removed November 24, 1928 and a like amount on December 12, 1928. At the present time, December 22, 1928, the abdomen is again much enlarged. No therapy other than liver feeding and rest was instituted. The use of novarsurol was considered but not instituted. The fluid output was much in excess of the intake during the early part of the patient's treatment.

*Laboratory Examinations*—Blood Examinations

- 11/ 2/28 Total red cells per cubic millimeter, 990,000 Hemoglobin, 40 per cent  
 Total white cells per cubic millimeter 6250 Color index, 2.2  
 Differential count S M 50, L M 0, E 0, B 0, N 50
- 11/ 6/28 \* Total red cells per cubic millimeter 1,420,000 Hemoglobin 41 per cent or 5.82 gm  
 Hematocrit volume 18.5 per cent  
 Average corpuscular volume 130.3 cubic microns  
 Average corpuscular hemoglobin 41 x 10  
 Percentage of hemoglobin in average cell 31.5 per cent  
 Volume index 1.59 Color index 1.43 Saturation index 0.9  
 Price Jones count average of 130 cells, 8.7 microns  
 Numerous megaloblasts, some normoblasts, reticulocytes  
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**Discussion**—Some observers consider pernicious anemia to be a rare disease in the typical negro. This is not so in my own experience.\* I believe it to be at least as common as in the white race. Ascites is said to occur with pernicious anemia, and probably small accumulations are common as part of the edema, so frequent in all severe anemias, but large collections of fluid in the peritoneal cavity, without accompanying edema, are rare. This man presents a large accumulation, and it was regarded, at first as merely part of the picture of pernicious anemia, but, though the blood rapidly became normal with the feeding of liver and liver extract, the ascites persisted and returned in just as large amounts following removal by paracentesis, the last tap was performed after the blood-picture was normal, yet the fluid returned just as promptly and in just as large amounts, varying in no way from the same phenomenon following the first tap, done when the anemia was at its worst. Palpation of the abdomen immediately after removal of fluid did not discover any masses, tumors, or enlarged organs. A pneumoperitoneum was attempted, but was unsuccessful. Cirrhosis of the liver seems to me to be the most satisfactory explanation of the persistent and recurring ascites, if this is the case it is interesting to speculate on the possible connection with the blood dyscrasia. Except for the presence of ascites, the general condition of the patient at the present time (December 22, 1928) is very good.

\* Jamison, C. Pernicious Anemia in the North American Negro, Southern Med Jour, 19, 583-584, August 26th

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cure Within recent years it has been found that iodoquinolin sulphonic acid possesses unusual amebicidal properties and is said to be remarkably free from toxic effects upon the patient It has been employed extensively by the German physicians under the trade name of Yatren, and it is manufactured in the United States by the Ernst Bischoff Company under the trade name of Anayodin The use of this drug in several types of amebic infections may be illustrated by the following cases, which have been selected as typifying the usual range of severity in clinical symptoms that is to be met with in amebic infections For brevity, only those symptoms pertinent to an amebic infection will be enumerated

Case I—Mr B, white, aged thirty seven years, was referred for diagnosis of a possible amebic infection by Dr John B Elliott, of New Orleans The patient's history recounted numerous attacks of dysentery during the past few months, accompanied by the usual symptoms of abdominal pains, tenesmus, and the passage of bloody mucoid stools There had been considerable loss in weight and he was practically incapacitated by general muscular weakness There had been an average of ten to twelve stools per day for the week prior to examination

The patient was requested to evacuate the bowels and the passage consisted almost entirely of mucus mixed with blood and was practically without fecal material Numerous small grayish particles of necrotic material were noted, which, when fished out and examined, proved to be largely masses of amebæ, as many as several hundred to each 16 mm field of the microscope being found Under higher magnification the amebæ were found to possess large clear pseudopodia, barely visible nuclei and numerous phagocytized erythrocytes with an absence of ingested bacteria The characteristic morphologic features of the amebæ, with the history of the patient, confirmed the diagnosis of infection with *Endameba histolytica* Proctoscopic examination was not possible, the ulcerations apparently extending to and involving the internal sphincter

Treatment—Three grams of anayodin (four 0.25 gm pills given three times daily before meals) were administered daily for a period of seven days A rest of five days was allowed and another seven-day course of anayodin given

By the third day of treatment all distressing symptoms had subsided and the number of stools dropped to three or four soft or watery fecal movements There were no vegetative or encysted forms of amebæ found after this time During the second course of anayodin the stools again became rather frequent, but the passages were without discomfort and did not contain blood, pus, or mucus At the end of the second week the patient had gained sufficient strength to resume work as cashier of a bank

Stool examination one month later showed a formed stool with no ab

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intestinal tract was negative. The leukocyte count was normal and there was no pus found in the urine.

Examination of a hard formed stool showed an enormous number of cysts of *Endameba histolytica* each containing from one to four nuclei. These were definitely identified by wet-fixed and stained smears. No history of dysentery or loose bowels at any time for the past few years was obtainable, in fact the patient had found it necessary to take daily enemas to evacuate the bowels.

Treatment with anayodin, 1 gm. three times daily for ten days, followed by a five-day rest, and continuing with a seven-day course of medication, resulted in a return of the temperature to normal and disappearance of the abdominal discomfort. I was not able to demonstrate amebic cysts after the fourth day of treatment. Monthly examination of feces for the past fourteen months has failed to show any return of the infection. The liver is still enlarged, but there has been no temperature and the patient feels in the best of health.

It is interesting to note that a brother of this patient was operated upon for a liver abscess some fourteen months previously. The contents of this abscess were sterile, of a chocolate color, and amebæ were found in the drainage fluid on the third day following operation. There had been no dysentery preceding this abscess, and no vegetative or encysted amebæ were ever found in the stools.

Case IV—Mr. J. K. D., a medical student, twenty-four years of age, apparently in good health, was found in the course of a routine class study to have numerous amebic cysts in his feces. These cysts were definitely identified as *Endameba histolytica*. He recalled having had an occasional attack of abdominal discomfort followed by a slight disturbance of the bowels for the past few months and which had been attributed to slight indiscretions in diet.

Treatment with anayodin, 1 gm. three times daily for seven days, followed by a five-day rest and continuing with another seven day course of treatment resulted in prompt disappearance of the cysts. Frequent stool examinations during the past two years have failed to show any recurrence of the infection, together with complete freedom from intestinal disturbances.

## DISCUSSION

**Symptomatology**—You are already familiar with the classical symptomatology of amebic dysentery, as shown by the first case presented, but I should like to call your attention particularly to the great variation, both as to the severity and as to the type of symptoms that may be exhibited in amebic infections in general. The symptoms in these patients have ranged all the way from a very debilitating disease produced by extremely extensive ulcerations of the colon with frank dysenteric symptoms, on through the mulder types without dysentery, types such as

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able to find It would appear that they all have from time to time some symptoms referable to actual tissue invasion of the colonic mucosa It is very doubtful if pathogenic amebæ can live and reproduce to any extent or for any length of time in normal fecal contents

All persons found infected with the pathogenic variety should be cured of their infection, for not only are such ameba carriers responsible for the spread of the disease, but those infected are themselves, at some subsequent time, apt to develop either dysentery or amebic liver abscess I have in my records one such case, a patient that developed an abscess of the liver eight months following the demonstration of cysts of *Endameba histolytica* in his feces Three months following the surgical drainage of the abscess the patient developed an acute amebic dysentery, and amebæ were found in the feces, as well as in the discharge from the abscess which had continued to suppurate and drain in spite of the efforts of the surgeon to sterilize the cavity

Diagnosis—A presumptive clinical diagnosis of amebic dysentery may be made and this confirmed by the finding of vegetative *Endameba histolytica* in the feces, but the diagnosis of the chronic, subacute, and carrier types of the infection is more apt to be made from routine stool examinations

In the presence of dysentery, search should be made for the motile vegetative forms immediately after passage of the stool, or preferably in material scraped from the rectal ulcerations Amebas are occasionally rather few in number, particularly in liquid stools, and with practice one may save considerable time and occasionally save a mistake in diagnosis by covering a comparatively large quantity of material with low magnifications

In the use of the proctoscope, the fact should not be overlooked that often the lesions are limited to the rectum, and particular search should be made for small ulcers just above the sphincter

In soft stools with precystic vegetative amebæ, these may be identified by wet-fixed smears stained with iron-alum-hematoxylin, or more easily by the characteristic cysts that will be

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Treatment of amebic infection should be directed toward an eradication of the infection and with this end in view no matter what form of treatment may be employed the patient should be carefully examined at intervals for many months

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"I saw this patient on August 30, 1927, he being referred to me for consultation by Dr W A Love At that time he gave me the history that while going downstairs he slipped and fell, hitting his left knee Local treatment did not help much Patient states that the pain is worse at night and does not radiate History of having fallen about eleven years ago striking his left tibia, since then parents have noticed that tibia began to enlarge and child has limped ever since

*"Examination*—Very thin, undernourished child, stands with both knees slightly flexed, feet in good shape except that anterior arches are relieved Teeth in fair condition, tonsils enlarged, but not reddened or inflamed, glands not palpable Typical sabre tibia, both legs Slight limitation of hip joint motion probably due to abnormal shape of head of femur Circumference left knee  $10\frac{1}{2}$  inches, right,  $10\frac{1}{2}$  inches

*"Skiaograph* which was sent by Dr Henriques shows marked osteoperiostitis of the right femur throughout its entire length even to the lower epiphysis Same condition in left femur, but to a much less degree, also in both tibiae and fibulas, the left tibia showing much more marked trouble than the right

*"My diagnosis* was congenital lues plus traumatism of a diseased left knee joint with synovitis I advised absolute rest and mixed treatment Since then I have not seen the patient "

A month from the date of the fall the patient was sent to our genito urinary dispensary with the request that he be given salvarsan The left knee was still swollen and stiff, slightly red and tender to touch In the dispensary he received five doses of salvarsan (0.2 gm) intravenously between October 4, 1927 and November 1, 1927 After two or three doses the swelling disappeared from his left knee and it was no longer stiff At this time his ankles were not swollen and he was not short of breath The day after his last dose of salvarsan he began to have smothering spells which he described as follows "My chest was tight and I could hardly breathe I would become short of breath, feel dizzy, and have a headache I had never previously had any similar attacks " These smothering spells would last an hour or two, occurring once or twice a night but never in the daytime when he is about sitting up helps to relieve the attack There has been no pain in the chest, merely a tight feeling with these smothering spells At times he expectorates a small amount of mucus with the hacking cough he has There has been no hemoptysis Exertion easily causes dyspnea for the past week His appetite is poor Digestion is good No nausea No vomiting He has a sour taste in his mouth Bowels regular Micturition  $\frac{1-2 \times N}{3-4 \times D}$  no burning His occupation requires him to be outside most of the time, exposed to the weather He does not smoke

*Past History*—Other than measles and mumps and an injury at the age of five years, he has never been sick He felt well up to the time of injuring the left knee two months ago

*Family History*—Father living, very nervous and has "rheumatism" His mother has been treated at another hospital for cerebrospinal syphilis,

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The tentative diagnosis, therefore, based upon this history and physical examination is

- 1 Infantilism unknown etiology
- 2 Luetic bone disease
- 3 Acute nephritis following administration of salvarsan

We are at a loss, however, to understand the attacks of dyspnea. His account of them suggested rather a bronchial asthma than an asthma dependent upon his nephritis. His infantilism challenged our interest and attention as all such cases do. It is the generally accepted, though very vaguely understood, idea that infantilism represents some endocrine dystrophy. Many of the cases of infantilism have been associated with definite evidence of disease of the hypophysis. We observe neighborhood symptoms, such as headache, evidence of pressure upon the optic nerves producing constriction of the visual fields or even total blindness. Some, x-ray pictures of the skull show abnormalities of the sella turcica and I have in several cases seen dense shadows due apparently to calcification or tumor mass in the hypophysis itself. In other cases there have been no such clear indication of hypophyseal disease and writers have suggested a disturbance of the internal secretion of the thyroid or of the gonads. Even if we accept an endocrinopathy as the basis of the pathogenesis of infantilism, it remains always to find the etiologic factor of the endocrinopathy. This undoubtedly is not always the same. Sometimes, as I have already indicated, the cause is a neoplasm. In another place, in studying the type of infantilism of hookworm disease, I have suggested that this type of infantilism is produced by the deleterious action of the hookworm toxin upon the endocrine system. In the case of our present patient who seemed to have a bone disease and perforation of the nasal septum due to hereditary lues, we were inclined to interpret his infantilism as due to an endocrinopathy caused by lues. This assumption, however, was proved unwarranted in the light of further investigation.

We first looked for any other evidence of pituitary disease. Dr H N Blum, oculist, reported the eye-grounds normal. The visual fields showed some concentric narrowing for color. Glucose tolerance tests were as follows

#### 100 gm of glucose given

##### Blood

	Mg per 100 c.c
Fasting specimen	58.8
$\frac{1}{2}$ hour after meal	95
1 hour after meal	91
2 hours after meal	68
3 hours after meal	91

#### One week later 100 gm of glucose given

	Mg per 100 c.c
Fasting specimen	76
$\frac{1}{2}$ hour after meal	143
1 hour after meal	110
2 hours after meal	100
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has made a special study of radiologic evidence of the development of the skeleton. Dr Henderson reported that the radiograms of the long bones exhibited the osteal development of a child between ten and twelve years of age. He remarked that there were certain bone changes which were suggestive of lues but they did not warrant this conclusion absolutely. Dr Isidore Cohn reported "Examination of the available plates which are those of the shoulder (Fig 207) and wrist (Fig 208) suggests that the child is about ten years old from the point of view of the skeletal development. This partial conclusion is arrived at because of complete ossification of the pisiform bone, the absence of the acromion apophysis and the wide epiphyseal line at the upper end of the humerus. I suggest pictures of the ankle, elbow, and hip as I believe conclusive evidence will be obtained from the presence or absence of the epiphysis of the olecranon, of the epiphysis of the os calcis, and of the epiphysis of the lesser trochanter." When these further skiagrams were taken in compliance with Dr Cohn's advice, Dr Henderson reported

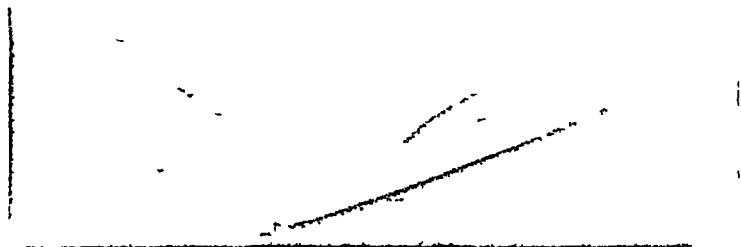


Fig 208 —Skiagram showing retarded development

"All the bones of the pelvis, lower extremities (Fig 209) and elbow region (Fig 210) show the status of development of the skeleton to be eleven years of age. The ilium has not yet fused with the ischium, the lesser trochanters are still free as is the olecranon process. A point of unusual interest observed in the left femur is a very extensive involvement in which the cortex is both expanded and thinned down with large cyst-like bodies observed in the medullary canal (Fig 210). Perpendicular striations are visible in the bone but no production has occurred beyond the periosteum. The changes are typically those seen in osteitis fibrosa cystica."

Meanwhile Dr H. L. Kearney had reported "There is a perforation of the septum involving only the quadrangular cartilage—the bony septum is not affected. This could be from lues but there is usually bone involvement in lues and a diagnosis of lues from the lesion alone is not warranted."

With these reports before us, we were confronted with a set of facts which permitted, if indeed it did not demand, an inter-

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Fig 210 —Skiergram showing retarded development, also the osteitis fibrosa cystica in the left femur



Fig 211 —Skiergram showing retarded development

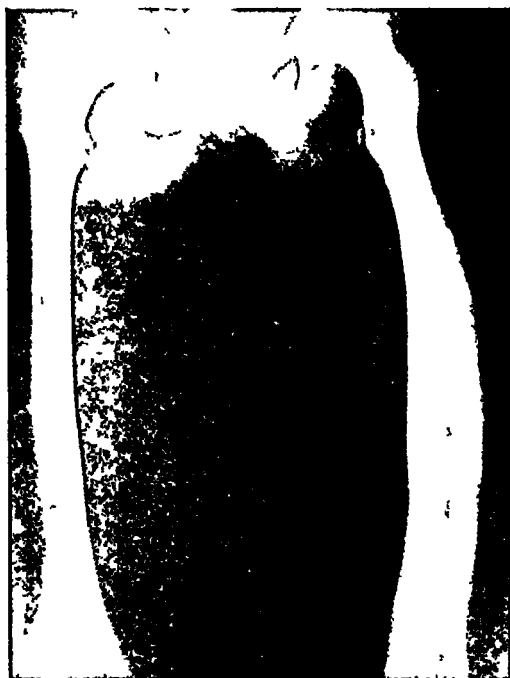


Fig 210 —Skiergram showing retarded development, also the osteitis fibrosa cystica in the left femur



Fig 211 —Skiergram showing retarded development



first osteoid but may become calcified so that the bone regains its rigidity "

There have been many suggestions that the two diseases—Paget's osteitis deformans and von Recklinghausen's osteitis fibrose oder deformirende (osteitis fibrosa solida or cystica)—are different forms of the same disease. The original conception was that von Recklinghausen's disease attacks children and young adults while Paget considered his disease one of mature life. "Seth Hirsch, Sikes, Packard, Stelle, and Kirkbride found in the study of 51 cases, the average age to be forty-nine and a half years"<sup>16</sup> The changes of osteitis fibrosa cystica have been found even in the early weeks of life. Ireland<sup>17</sup> reported the youngest case on record—a child forty days' old. I. Seth Hirsch<sup>16</sup> believes that the disease is probably congenital with strong hereditary tendencies.

Cysts are frequent in osteitis fibrosa and relatively rare in osteitis deformans, although they do occur in the latter disease. Many have pointed out that the histologic pictures present transitional forms between the two diseases. While it cannot be said that they are surely identical, there is considerable evidence suggesting this. Thus Eising<sup>6</sup> "Although osteitis deformans and osteitis fibrosa present decided clinical differences, upon histologic analysis, these differences are more in quantitative ratio than in fact." There is neither time nor purpose to elaborate here upon this point. I wish merely to indicate that there is probably underlying these two diseases, and probably also the single bone cysts, the benign giant-cell tumors, and similar bone diseases, a fundamental constitutional depravity. I do not mean by this that the basic condition is necessarily the same in all of the bone dystrophies. As Landon<sup>7</sup> points out, no sharp distinction can be made between osteitis fibrosa and the solitary cysts. The solitary cyst is limited to *one* bone in which the cyst formation is the prominent change, while in general osteitis fibrosa cyst formation is of minor importance. The essential underlying process leading to the production of cysts is osteitis fibrosa of the type limited to a part or whole of a single bone.

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in 2, 3 had sclerotic thyroids. Many reports make no mention whatever of the glands of internal secretion.

"Da Costa interprets the retention of calcium, phosphorus, and magnesium, with the sulphur loss found in these cases, as indicating a stimulated osseous or osteoid formation, accompanying the resorption of a highly sulphurized organic matrix. In the course of this calcification procedure, he supposes that a certain quota of the sulphur of the matrix is replaced by other elements, a process which must entail retention of calcium, phosphorus, and magnesium, and increased elimination of sulphur. He shows the close parallelism between the mineral metabolism of a growing boy, a case after parathyroidectomy, and a case of osteitis deformans, and suggests that this depends in some way, either on the absence or on the perversion of some internal secretion, possibly the parathyroids, which controls calcium exchange in the body. From some cause, substances arise which have the power to abstract calcium from the body tissues, the abstraction of these salts being the first step in the production of the disease. This theory of Da Costa's most satisfactorily describes the history of the monkeys which we wish to report."

The rôle of the parathyroid in regulating calcium metabolism has been made clearer in recent years since the isolation of its hormone by Collip. A suggestive point is that there are in the literature a number of reports of osteitis fibrosa cystica associated with parathyroid tumors. Such are the cases of Gold,<sup>10</sup> Godel,<sup>11</sup> Meyer,<sup>12</sup> and Dawson and Struthers.<sup>5</sup> Meyer cites similar findings of Schmorl, Erdheim, Askanazy, Strada, and Bower. In our present patient we have found no evidence of parathyroid tumor. His blood-serum calcium was 11.2 mg per 100 cc—a perfectly normal finding. Liles and I<sup>13</sup> have pointed out elsewhere in connection with the loss of bones in leprosy that gradual resorption of bone tissue is not accompanied by any disturbance of the blood-serum calcium level nor was such disturbance to be expected in view of the physiologic regulatory mechanism. To have more definite information upon this point, it would be necessary to study the calcium phosphorus magnesium sulphur balance as White did in his monkeys.

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*Physical Examination*—Healthy looking, obese woman Weight 178½ pounds Height 5 feet, 7½ inches Skin Normal No adenopathy  
Mucous membranes Normal Throat Negative Tongue Somewhat coated Neck Negative

Thorax 97 cm, 102 cm full inspiration, 75 cm full expiration No tenderness on percussion over the sternum

Lungs Normal

Heart Rate 88 sitting Blood-pressure 126/70

Dulness fifth left intercostal space 8 cm from the midline

Dulness third right intercostal space 1 cm from the midline

No murmur No irregularity

Abdomen Relaxed, tenderness in the right hypochondrium Liver and spleen not felt

Knee-jerks Normal

Scar over the frontal eminence on the right side Suggestion of external strabismus of the right eye

Pupils Equal, regular, symmetrical, react to light and accommodation

*Fluoroscopic Examination*—Heart Normal

Aorta Normal In the posterior mediastinum an indefinite shadow about halfway down

Lungs Normal

*Laboratory reports*

Urine A M 1022 acid yellow turbid albumen trace, sugar 0, indican 0

P M 1024 acid yellow turbid albumen heavy trace, sugar 0, indican 0

Aldehyd 0

Sediment Pus large amount, mucus, ep cells, urates

Blood Wassermann negative

Hemoglobin 85 per cent No abnormal cells found in smears

Thinking perhaps that her troubles were due to gall-bladder disease I sent her to Dr W F Henderson with the request that he try to obtain a visualization of the gall-bladder after the administration of dye He reported as follows

"Following the administration of the opaque dye, the gall bladder shadow is observed to begin filling upon plate No 1, plates No 2 and 3 show an increase in filling and concentration, while plate No 4 shows a satisfactory emptying and the evidence here obtained is insufficient to condemn this gall-bladder At fluoroscopic examination of the chest, the cardiac shadow appears to be normal in size but the great vessels are somewhat widened, particularly just above the heart In the lateral projection the aorta appears to be somewhat wider than normal The administration of the barium capsule shows this to come to rest in the posterior mediastinum (Fig 212), behind the upper margin of the heart, remaining at this location over a considerable period of time Following the administration of four doses of atropin the procedure is repeated Again the barium capsule comes to rest in the identical location As the capsule breaks up and the barium proceeds slowly through the constricted portion of the esophagus, the esophageal wall does not present the cauliflower-like projections and irregularities of its margins which are so commonly seen in malignancy, and it is impos-

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She then went home but returned in about a month saying that she had not been able to eat any hard food. She took only liquids and soft cereals. She felt weak but had actually gained weight. On this second visit, Dr Weil and Dr H. L. Kerney did an esophagoscopy again, September 16, 1927, and reported

"Nine mm full lumen Jackson-Moshier esophagoscope passed into stomach without encountering any evidence of esophageal obstruction. Inasmuch as the fluoroscopic examination had found evidence of esophageal obstruction



Fig 213 — Anteroposterior view of chest, August 11, 1927

at about the level of the crossing of left bronchus, this neighborhood was very carefully examined for pathology or pressure. There was no evidence of encroachment on the esophageal lumen either by endo esophageal pathology or by pressure from some external structure. On the anterior esophageal wall, 26 cm from the upper incisor teeth was encountered an area of mucosa about 8 mm in diameter which did not look normal, there seemed to be some slight thickening in this area."

She went on a full diet at the end of the week when the soreness of her throat had subsided. She ate with relish and without difficulty until October 8th when after eating peanuts she experienced the same kind of pain behind the sternum that she had originally had, only less intense

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Fig 215 —Lateral view October 15, 1928 showing the mass still present It is as large as ever, but apparently not as dense

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The tissue from the bronchus was reported on by Dr J A Lanford, pathologist, as follows "The tissue is a polypoid like structure made up of supportive tissue containing blood vessels and showing a number of lymphoid and plasma cells scattered throughout, they being particularly striking around the blood vessels The surface is covered with stratified squamous epithelium which is adult in type, with the

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April 4th the sputum was still stained with blood On April 12th she brought with her in a little vial the polyp which she had spat up Dr Lanford reported on this as follows

"Examination of the tissue shows it to be a polypus which just above its attachment is covered with a few epithelial cells, some of which are ciliated, but for the most part the surface is free of epithelium The polypus is made up largely of blood-vessels, very loose connective tissue and inflammatory reaction, both acute exudative and chronic proliferative It gives a picture of granulation tissue and nowhere is there any suggestion of a malignant neoplasm "

She had had no retrosternal pain except in February and again on April 3d She looked fine Physical examination revealed nothing of importance

July 9, 1928 Except for a cough she did very well until the first week in June when she had abdominal pain and loose bowels for a few days Following this she lost her appetite and has not recovered it About June 18th she began having retrosternal and interscapular pain after swallowing Because of this she took only soft food for two or three days and as this did not make the pain any better she went on a diet of malted milk only The pain then stopped and when she resumed eating it did not return She still had a sensation of a lump or soft rubber ball retrosternally or retromanubrially Slight cough, but no odor to the breath now She did have an odor to the breath with the cough the middle of June Although she thought she had been losing considerable weight since about the middle of June, she still weighed 183 pounds Physical examination revealed nothing of importance. x Ray report July 9th was as follows

"In the anteroposterior position the diaphragms, heart, and aorta appear to be normal and while the structures at the hilum are somewhat wide, they are not otherwise characterized by evidence of pathology In the lateral projection the chest appears to be normal with the exception of the shadow which was previously observed in the posterior mediastinum and is still exhibited as a large oval mass behind the aorta and auricular region of the heart This shadow is still smooth but appears to be considerably larger than at the previous examination The shadow can be likened to the size of a very large gall-bladder "

July 11, 1928 "Bronchoscopy was done with the 7 mm Jackson aspirating bronchoscope and revealed a stenosis of the right bronchus, about an inch beyond the bifurcation This was apparently due to an encroachment on the lumen by thickening of the bronchial wall on the left side at the site of the polypus, which was removed last year Bronchial motility at this point was normal There was no infiltration suggestive of malignancy Esophagoscopy was done with the 9 mm Jackson full lumen esophagoscope, stomach was entered without difficulty The area previously noted 26 cm from the incisor teeth did not display any evidence of pathology "

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new growth, and after the lapse of a year a new growth which had metastasized to the mediastinum would have certainly itself become evident and would have by this time made the patient cachectic. This she is not. Secondly we may think of the lymph-nodes being enlarged in the course of some blood dyscrasia. This hypothesis is negatived at once by the result of the blood examination. Thirdly, the lymph-nodes may be the site of tuberculosis. This cannot be regarded as probable in the absence of any evidence of tuberculosis in the lungs. Fourthly, the lymph-nodes may be enlarged as the result of syphilis. This cannot be regarded as probable as there is no other evidence



Fig 215a—Lateral view of chest, December 16, 1928, showing complete disappearance of the mass in the posterior mediastinum

in the physical examination and history or the Wassermann reaction of the patient. Finally, the lymph-nodes in the mediastinum may be enlarged as the result of infection of the polyp in the bronchus and the subsequent drainage to the lymph-nodes. This by exclusion seems to be the most plausible explanation and it is the one that we have adopted as the tentative and working diagnosis. I confess that it is open to objection—first that the mass is unusually large for lymph-nodes and second that with the removal of the polyp there should have been some recession of the glandular infection and enlargement.

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*Physical Examination* —A small, white man, well developed, and fairly well nourished. He is orthopneic and cyanotic. He appears restless but complains of no pain. Temperature 101° F, pulse 130, regular, respiration rapid. Slight edema of ankles and feet. Moist râles audible at both bases. Heart is enlarged slightly to the right but chiefly to the left where it extends to the anterior axillary line in the sixth intercostal space. At the apex the first sound is distant and muffled, the second sound is louder. At the junction of the fifth rib and left sternal border is heard a to and fro murmur, the sounds are creaking and leathery in character and quite loud. This rub is present when the breath is held (patient complains of no pain). Liver border is palpable. Blood-pressure 135/95.

Urine Specific gravity 1.020, otherwise negative.

Phenolsulphonephthalein test 35 per cent in two hours (intravenously).

	Per 100 c c blood
Total non-protein nitrogen	49.8
Urea nitrogen	27
Uric acid	4
Creatinin	1.57
Dextrose	105

Red blood cells 3,500,000, hemoglobin 75 per cent, leukocytes 7500, neutrophils 82 per cent, small lymphocytes 18 per cent. No red cell changes, no plasmodia.

Wassermann negative.

June 19th Pericardial rub has disappeared.

June 22d Much improved. No fever for past two days. Edema of feet and ankles gone. Blood-pressure 105/80.

June 23d Blood-pressure 118/90.

Phenolsulphonephthalein test 50 per cent in two hours (intravenously).

June 27, 1925 Patient discharged against my advice. No dyspnea or edema. Lungs clear. Heart smaller on percussion and cardiac sounds are louder. No murmurs. Weight 108 pounds.

*Comment* —Patient admitted with orthopnea but very slight edema of feet. Complained of no pain but had a temperature of 101° F, a well marked pericardial rub associated with an enlarged heart. Blood count on admission showed only 7500 leukocytes but with 82 per cent neutrophils. Blood pressure was 135/95. The symptoms and signs were suggestive of coronary occlusion, and it is unfortunate that no further blood counts were made. The rub disappeared on the third day and the fever fell gradually to normal in five days. There was also a falling blood pressure to 105/80, which gradually increased prior to discharge. In spite of the evidence of coronary disease it was thought advisable to administer digitalis in view of the evidence of congestive failure. He received a total of 10½ drams of tincture of digitalis in seven days and made a prompt recovery. The maximum diuresis in twenty-four hours was 56 ounces. The patient, upon discharge, was advised to take a prolonged rest which he did not do.

Second admission November 14, 1925 Discharged December 2, 1925

*Physical Examination*—A small, white man, well developed, and fairly well nourished. He is orthopneic and cyanotic. He appears restless but complains of no pain. Temperature 101° F, pulse 130, regular, respiration rapid. Slight edema of ankles and feet. Moist râles audible at both bases. Heart is enlarged slightly to the right but chiefly to the left where it extends to the anterior axillary line in the sixth intercostal space. At the apex the first sound is distant and muffled, the second sound is louder. At the junction of the fifth rib and left sternal border is heard a to-and-fro murmur, the sounds are creaking and leathery in character and quite loud. This rub is present when the breath is held (patient complains of no pain). Liver border is palpable. Blood-pressure 135/95.

Urine Specific gravity 1.020, otherwise negative.

Phenolsulphonephthalein test 35 per cent in two hours (intravenously).

	Per 100 c c blood
Total non-protein nitrogen	49.8
Urea nitrogen	27
Uric acid	4
Creatinin	1.57
Dextrose	105

Red blood cells 3,500,000, hemoglobin 75 per cent, leukocytes 7500, neutrophils 82 per cent, small lymphocytes 18 per cent. No red cell changes, no plasmodia.

Wassermann negative.

June 19th Pericardial rub has disappeared.

June 22d Much improved. No fever for past two days. Edema of feet and ankles gone. Blood-pressure 105/80.

June 23d Blood-pressure 118/90.

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Second admission November 14, 1925. Discharged December 2, 1925.

of digitalis in six days, then 30 minims daily until discharge. There was obviously no need for further medication.

Third admission, January 15, 1926 Discharged May 1, 1926

Patient returned to the hospital with congestive heart-failure more marked than on previous admissions. During his long stay in the hospital he showed evidence of decompensation on two occasions due to constantly getting out of bed and walking about against orders. It was impossible to control this patient satis-

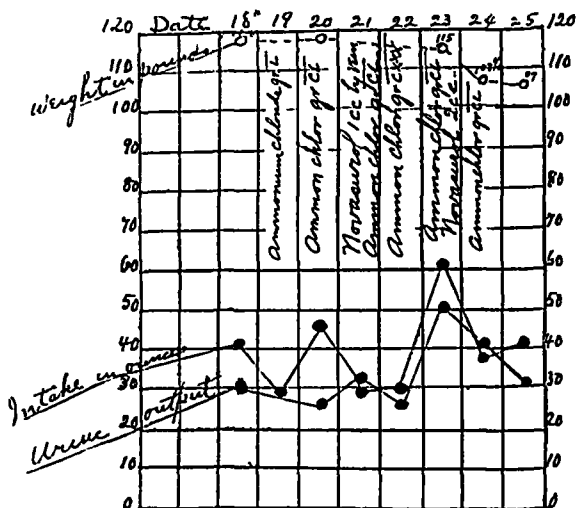


Fig 217—Third admission Patient digitalized Loss of weight due to toxic gastro enteritis from novasurol poisoning Diuresis slight

factorily because of his low order of intelligence. In these periods of decompensation, digitalis for the first time failed to entirely clear the edema or produce marked diuresis, in spite of the fact that full doses were employed. In view of this fact, resort was had to other diuretics.

Figure 217 shows the result following the use of ammonium chlorid and novasurol. The first dose of 1 c c of novasurol by vein administered on the third day of the ammonium chlorid therapy failed to produce any diuresis. He was given a second dose of 2 c c of novasurol two days later. This caused some

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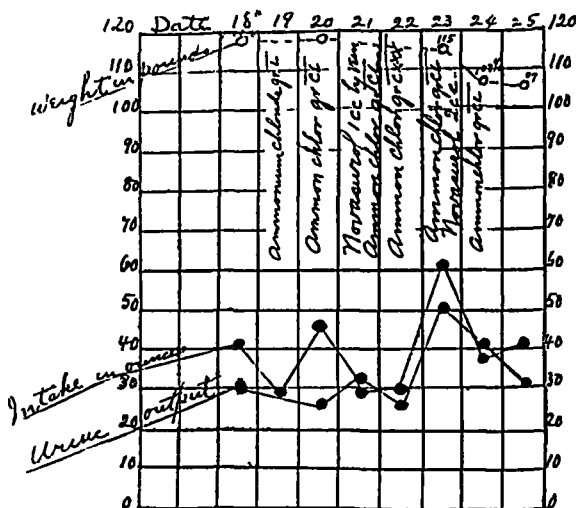


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evidence of myocardial damage by the presence of an alternation of the pulse. The liver was two fingerbreadths below the costal margin. Abdomen distended. Edema of feet and ankles. In this admission full digitalization again failed to abolish the edema. In addition to digitalis therapy, urea was administered from November 9th to 26th inclusive, without any appreciable diuresis or diminution of edema. In extenuation for the failure of urea, it should be noted that the dose was only 24 gms. a day. Considering the patient's comparatively low blood-urea the dose might well have been twice that amount.

On December 11th theocin was given to this patient for the first time. Figure 218 shows that his weight had increased to 126 pounds on December 10th. After four days of theocin in doses varying from 5 to 15 grains per day, there was a loss of almost 16 pounds. On the second day of theocin administration, the chart shows an output of almost 70 ounces in the twenty-four hours. This does not represent the total output as several specimens of urine were lost. The diuretic effect of this drug was truly remarkable. The edema disappeared entirely and his general condition showed marked improvement. Upon discharge he was again strongly advised to continue his digitalis.

Fifth admission February 7, 1927. Discharged March 22, 1927.

Patient's condition on this admission was somewhat better than previous one as he had continued his digitalis (much to our surprise). His chief complaints were shortness of breath and swelling of his feet.

Physical examination revealed moderate dyspnea and cyanosis. Pulse 96, regular. Moderate edema of feet and sacrum. Edema of bases of lungs. On this occasion he was put to bed as usual and the digitalis dosage increased still further until the electrocardiographic curves showed digitalis block and pulse slowed down to between 70 and 80 per minute, without abolishing the edema. Then theocin was given in addition.

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**Discussion**—It is not my purpose in the report of the above case to laud the remarkable action of theocin at the expense of other valuable diuretic agents. Novasurol (or merbaphen) has

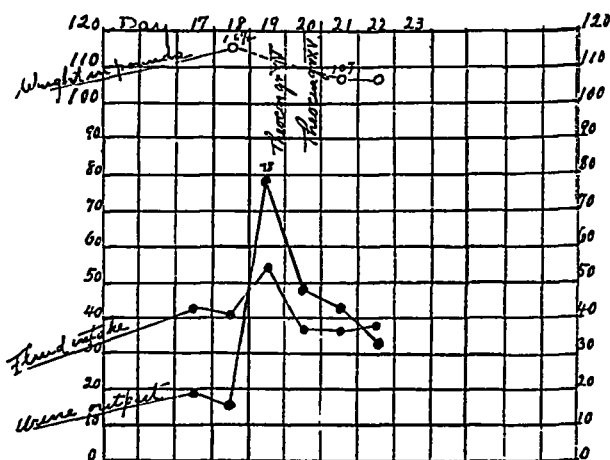


Fig 220—Sixth admission. Patient digitalized, but showing evidence of congestive heart failure.

proved its worth in many noteworthy instances. I have chosen this case as an introduction to a few brief remarks as to the status of certain diuretics in the treatment of cardiac edema.

Almost all clinicians agree that the edema associated with circulatory failure is usually best controlled and eliminated by thorough digitalization. There are, however, a small proportion of cases in which, in spite of full digitalization, rest, diet, and other measures, the edema does not entirely disappear. This condition of affairs most frequently presents itself in the cardiac patient who has had repeated attacks of congestive heart-

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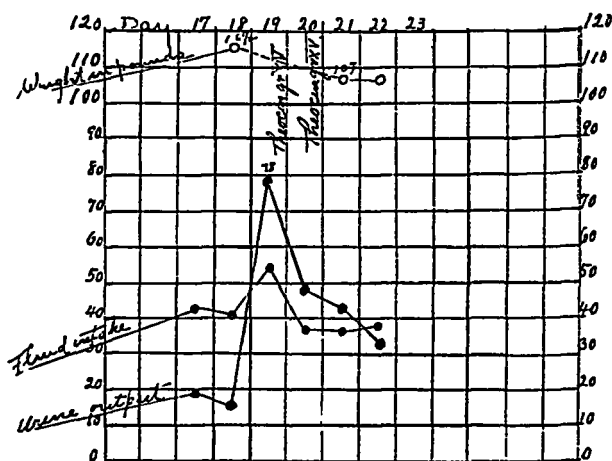


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prising, at times, to see the response of even badly diseased kidneys. It is also becoming clearer that the diuretic activity of these drugs does not depend entirely upon their action on the kidneys, but that there are also extra renal factors to be considered. The same drug will exhibit in one instance a remarkable diuretic effect and upon another occasion, under apparently the same conditions, will produce no increase in urinary output.

Experience has shown that novasurol is more effective when preceded by and given in conjunction with ammonium chlorid. Ammonium chlorid appears to pave the way for novasurol, possibly by altering the acid-base equilibrium of the body. Keith confirmed Haldane's observation that ammonium chlorid when taken in adequate amount diminishes the normal alkali reserve in the blood and tissues (produces an acidosis). In combination, ammonium chlorid and novasurol cause acidosis, an increase in the chlorid content of the blood and an increase in the excretion of water, chlorid, and fixed inorganic base, chiefly sodium, potassium, and ammonium. In the diuresis by theocin and digitalis there is an increase in chlorid, sodium, and fixed base similar to that produced by novasurol (Keith *et al*).

Stieglitz, in a recent illuminating paper, states that the paradox of diuresis by either acidification or alkalinization is open to logical physiologic interpretation. Any deviation from normal physiologic physical or chemical conditions, if the deviation is not so excessive as to be destructive, is a stimulant to activity on the part of the cells to return toward the normal environment. The significant fact is that either procedure, administration of acids or alkalis, causes a deviation from the pre-existing state and, therefore, acts as a renal stimulant. The result of renal stimulation is diuresis. Injured cells, already hyperirritable, are more readily influenced. In nephritis the acid renal cells are made more acid by the administration of alkalis and it is suggested that this added insult is in part responsible for the clinical diuresis so obtained. If the original injury is severe, the additional insult, instead of being stimulating, may be destructive or injurious, with acute exacerbation of the nephritic or toxic edema, as is so often the case. On the other hand, therapy with acid substances

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The stress laid by such writers as Lawrason Brown on the actual amount of blood in each case of blood-spitting is most important, and the insistence that blood spitting of a dram or more should be regarded as strongly probable evidence of tuberculosis is eminently correct. However, the practitioner who accepts such a dictum and rests his diagnosis upon it alone, failing to exhaust all diagnostic resources to prove his hypothesis, sometimes comes to grief. Repeated moderately large hemoptyses occurring in young subjects with mitral stenosis are sometimes confused with tuberculosis. This mistake while occasionally justifiable in atypical or latent cases, is, of course, inexcusable where the physical findings are perfectly typical of stenosis. A case in point was a strong looking young taxi driver who was

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The conclusion of the whole matter of hemoptysis or blood-spitting may be summed up about as follows. It is a serious thing for a person to expectorate blood and its source always should be sought. It is likely that nine-tenths of all hemoptyses of a dram or more come from diseases of the lungs and by far the most common disease is tuberculosis. Whether the blood results from actual invasion of the lung tissue or the rupture of a tuberculous bronchial gland into a bronchus is of speculative interest largely. It makes no difference that the hemorrhage occurs only once and there follows a long period of perfectly good health. We must insist on the seriousness of the symptom, although we congratulate the victim on the *apparent* healing of the lesion with complete restoration of his health. With what appears to be a greatly increasing frequency of neoplasm of the lungs and bronchi, we should keep this condition in mind in all cases of hemoptysis. By impressing upon our patients the seriousness of this single symptom we shall be able to keep them under observation for longer periods and thereby increase our percentage of correct diagnoses of many of the diseases in question.

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The patients of the group to which I invite your attention do not present the well-defined, easily recognized characteristics described in thyroid deficiency of high degree Sometimes their disability can best be described merely as "poor health" Before the true nature of their trouble is discovered they drift, as a rule, from doctor to doctor and are given various diagnoses Often they are called neurasthenics Nevertheless, there are certain disabilities which are common to all, and a few additional troubles which are manifested by the majority, all of which taken together give to the disorder a distinct identity

The salient characteristic is lack of endurance These people are unable to stand any sort of strain, physical or mental While they offer all sorts of complaints, to the observant physician it is evident that easy fatigue troubles them most, and chronic nervous exhaustion is a frequent diagnosis While this applies to mental as well as physical effort, there is no impairment of the patient's reasoning powers He merely is not capable of sustained mental effort and is lacking in initiative He is as a rule fully aware of this disability, and will oftentimes complain that he feels inadequate to meet his daily obligations

Numbness and vague pains in the legs and arms, particularly after exercise, are frequent To some patients this is very distressing Chronic joint pains occasionally occur

Vague digestive disturbances are often present and may constitute the chief clinical feature Constipation is frequent I first became interested in this group of patients when I learned that certain persons with vague gastro-intestinal disturbances, not characteristic of any definite disease, are occasionally benefited in a graphic manner by the administration of thyroid substance

A few men of this group will complain of loss of libido as the chief symptom, and in other instances inquiry will reveal definite deficiency in this regard In one of our cases this was the only complaint

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A low-grade secondary anemia is often present, but there is otherwise nothing noteworthy about the blood. The urine often contains albumin. Many of these patients are erroneously believed to have nephritis and are treated for this disease.

The basal metabolism is always low, and furnishes the final criterion by which the patient is judged. A rate which fails to reach the normal by as much as 10 per cent is ordinarily said to be abnormal, but this margin is probably too small. With the apparatus and technic in clinical use today it seems better to assume arbitrarily that  $-15$  per cent is the point at which the abnormal begins. All of the patients of our group have had a basal metabolism of 15 per cent or more below the calculated normal. Often it was  $-20$  to  $-30$  per cent.

Emphasis should be placed upon the necessity for care in estimating the metabolic rate. Complete rest of an hour or longer on a couch in a room alone before beginning the test is advisable. The mental state of the patient during the test is also important, if he is anxious, nervous, and frightened the curve is apt to be irregular and the result untrustworthy. The kymographic tracing in our cases is always scrutinized and unless it shows a fairly straight line indicating even breathing the test is discarded and another taken. We have usually found in such instances that after a few repetitions satisfactory results could be obtained, but occasionally we have encountered a patient who could never learn to take the test placidly and whose basal metabolism we could never accurately determine, these last are reminiscent of the soldiers who cannot wear a gas mask.

**Discussion**—The disabilities described are probably identical in nature with those of true myxedema, the difference being merely one of degree. However, the vagueness and mildness of the symptoms and the absence of the graphic skin and other changes seen in true myxedema entitle these patients to a separate grouping. An important difference is in the permanency of the disability. Myxedema represents thyroid deficiency of high degree which is permanent, but the disturbances of which I write are apparently dependent upon a mild degree of thyroid disorder which in many instances is of transient duration.

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Regarding the appropriate dosage of desiccated thyroid, Plummer administers in true myxedema a large initial dose, 30 to 60 grains (2-4 gm) which immediately gives a pronounced boost to metabolic activity. Then, after an interval of several days the drug is resumed in smaller doses. In the milder degrees of thyroid deficiency, however, such as are experienced by the patients of this group, it seems advisable to begin with smaller doses and to permit a more gradual effect. True, this takes time but under these circumstances it is probably safer. Usually a period of two or three weeks is required to accomplish the desired results, sometimes longer. Three or 4 grains of desiccated thyroid daily is appropriate in the beginning. Occasionally I have used double this amount for short periods with satisfactory results. At the end of about two weeks, sometimes longer, the effect of the drug makes itself evident, it is advisable then to reduce the dose to  $1\frac{1}{2}$  or 2 grains, and again after another two or three weeks to approximately 1 grain daily.

It is not always easy to determine the proper dosage of thyroid substance. The iodine assay of a thyroid preparation is taken as the measure of its potency, but experience has shown that not all standard preparations of thyroid substance on the market are of equal efficacy. It should be added, too, that all people do not absorb the drug with equal promptness and that in those cases where especially large doses appear to be necessary it is probable that the drug is not fully absorbed from the bowel. Changes in dosage will not be immediately felt, therefore, in the effort to properly adjust the dose any contemplated increase or decrease should not be too great, and at least a week or ten days should be allowed to elapse before judgment is taken as to the effect.

The patient's basal metabolism is after all the best guide to dosage. If feasible, frequent estimations of metabolism should be made during this early period of adjustment. Thereafter estimations should be made at longer intervals. Three or four weeks, sometimes longer, is required in the above described procedure to bring the basal metabolic rate to normal. After this has been

Regarding the appropriate dosage of desiccated thyroid, Plummer administers in true myxedema a large initial dose, 30 to 60 grains (2-4 gm) which immediately gives a pronounced boost to metabolic activity. Then, after an interval of several days the drug is resumed in smaller doses. In the milder degrees of thyroid deficiency, however, such as are experienced by the patients of this group, it seems advisable to begin with smaller doses and to permit a more gradual effect. True, this takes time but under these circumstances it is probably safer. Usually a period of two or three weeks is required to accomplish the desired results, sometimes longer. Three or 4 grains of desiccated thyroid daily is appropriate in the beginning. Occasionally I have used double this amount for short periods with satisfactory results. At the end of about two weeks, sometimes longer, the effect of the drug makes itself evident, it is advisable then to reduce the dose to  $1\frac{1}{2}$  or 2 grains, and again after another two or three weeks to approximately 1 grain daily.

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an existing deficiency, it is beneficial, but if given in excess of this amount, or when there is no deficiency, it is merely toxic. There may be a few exceptions, but it can be stated as a general rule that thyroid substance should not be given unless it is definitely known that the basal metabolism is below normal.

Thyroid substance should be given alone, never in combination with other glandular preparations. There are many polyglandular extracts in use, but fortunately most of them are inert except as regards their thyroid content. Polypharmacy is always bad and it is especially bad when used as a means for steering the ship through "uncharted seas." However, much we may surmise as to the intimacy of physiologic relationship between the several organs of internal secretion, we should endeavor in our therapeutic efforts to confine ourselves to what we actually know. Therefore, it is infinitely better always to give thyroid substance alone.

It should be reiterated that there is danger here of over-enthusiasm. Because thyroid substance benefits in a graphic manner a few patients who are deficient in thyroxin there is a temptation to prescribe it with little discrimination. Not every patient who is tired, or is lacking in enthusiasm, or who has a low pulse pressure, or a bottle-shaped heart, or who experiences loss of libido, is a sufferer from thyroid deficiency. The patients who are placed in this category and are given thyroid substance should be selected with care, preferably after metabolic determinations. There are few therapeutic measures which when needed bring more satisfactory results, and there are few which when improperly used are more clearly capable of harm.

### ILLUSTRATIVE CASES

Case I—F V, a business executive of forty nine years, has been under my care from time to time for about ten years because of occasional backache and frequent periods of extreme lassitude. Repeated examinations showed no cause for his troubles until it was found two years ago that he had a basal metabolic rate which varied between -17 per cent and -20 per cent. His pulse rate had always been low, about 60, and the blood pressure moderately low, 100/60. He was of approximately normal weight and his appearance except for a sallow skin has always been vigorous. Under thyroid medication, 2 grains of the desiccated powder daily at first and then 1

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in the central nervous system. It should be constantly remembered, however, that this disease limits itself to no single system or structure.

Before proceeding to the consideration of the case I wish to remind you that immunity is a factor in syphilitic disease as in other infections. In the untreated disease the patient's resistance is of prime importance.<sup>1</sup> Without the development of this resistance, which spontaneously terminates the secondary stage of the disease, the individual would probably succumb to an overwhelming, generalized treponemal infection just as the syphilitic fetus succumbs. Thanks to this natural force the body, unaided, usually checks the infection and passes into a period of latency or quiescence. This, it must be remembered, is an immunologic reaction capable of being modified by any factor interfering with the natural development of the disease, as anti-syphilitic treatment.

The case to be reported presented, when first seen, important diagnostic problems, but is of especial interest because it illustrates impressively the dangers incident to the haphazard, casual, inadequate treatment of early syphilis. That such treatment may operate to the detriment of the patient is illustrated in this case—the way in which it operates will be suggested in the discussion which follows the case report.

**Case Report**—The patient is a traveling salesman twenty five years of age and unmarried. He was brought to the hospital in an ambulance. He was unable to give a connected account of his troubles, and only an incomplete history was obtained from his mother.

Nine days before admission to the hospital he felt fairly well with the exception of slight headache. The latter had been present for some days but had not interfered with his work. On the evening of the ninth day before his admission he retired to his hotel room and went to bed. He complained to a friend on saying good night that his head had ached badly all day and he retired early for this reason. The next morning he was found on the floor of his room in a dazed state, disoriented as to time and place, and with no recollection of what had transpired during the night. There was an abrasion on the tongue and lip and a superficial scalp wound was present. These findings led the attending physician to the conclusion that he had had a generalized convulsive seizure and he was placed in a hospital. Here he remained for two days. During this time he was restless, irritable, and altogether incorrigible. He complained bitterly of persistent headache. On the

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suffered intensely from generalized headache which could only be temporarily and partially relieved. The skin and mucous membranes were normal except for the partially healed abrasions of the tongue, cheek, and scalp. A few shotty glands were palpable in the inguinal and posterior cervical regions. No generalized glandular enlargement was present. The remainder of the general physical examination revealed the following positive findings. The peripheral vessels were everywhere abnormally thickened and the aortic second sound had a distinctly ringing, metallic quality, the blood-pressure was 120/60, a soft, pigmented scar was present in the sulcus near the frenulum. The only positive findings on neurologic examination were distinct hyperemia of the retinae with fulness of the veins and indistinctness of the borders of the optic disks, though physiologic cupping was present. There were no signs of meningeal irritation except brisk, though not distinctively hyperactive reflexes. The usual examinations of the urine and blood revealed normal findings. The Wassermann reaction with the blood was negative with both alcoholic and cholesterinized antigens. The Kahn test for syphilis was doubtful, distinct precipitation occurring in only one of the three dilutions of antigen used. A lumbar puncture was performed on the day of admission. The cerebrospinal fluid was under definitely increased pressure and contained 80 cells per cubic millimeter. The test for globulin was strongly positive and the colloidal mastic test was positive, showing reaction No. 4 in one tube. The Wassermann reaction was positive with 1 and 0.5 c.c. of fluid.

The case may be summarized as follows. A man twenty-five years old, admitting exposure, developed a painless lesion on the penis which was considered non syphilitic. Approximately two months later a diagnosis of secondary skin syphilis was made, treatment instituted and continued until three injections of neo-arsphenamin had been received. At that time on the advice of another physician and because of the absence of clinical or serologic (blood) evidence of syphilis, treatment was discontinued without immediate harmful effect. Eight to ten weeks later headache, convulsive seizures, disorientation and mental confusion developed. Ophthalmoscopic examination revealed indistinct optic disks and congested retinae; the reflexes were brisk. A low fever was present. The cerebrospinal fluid examination gave conclusive evidence of syphilitic meningitis.

A diagnosis of syphilitic relapse or recurrence was made. The case clearly belonged to the "meningeal recurrence" or "meningorecidive" group. Antisyphilitic treatment resulted in a prompt symptomatic cure of the meningitis with return of the spinal fluid findings to normal after approximately 8 gm. of neo-arsphenamin and 7 grains of the salicylate of mercury. Intensive treatment was continued.

**Discussion**—In considering the diagnosis of this case one is impressed with the following fact. The signs and symptoms of clinical neurosyphilis developed within five months of the appearance of the chancre, and within eight or ten weeks of the last neo-arsphenamin treatment. As a rule clinical evidence of

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The above brief survey of the progress of the disease in an untreated case of syphilis is given in order to again emphasize the fact that a defensive mechanism operates in syphilis—that there develops a real though usually incomplete immunity to the disease. This defense mechanism rarely, if ever, completely eradicates the agents of the disease, when once they are entrenched. I know of no reported case of spontaneous cure of syphilis. However, it reduces the infection from an acute, generalized, fulminant one to a chronic, localized, quiescent one. As I have previously said, without this mechanism the untreated syphilitic would probably succumb to the generalized treponemal infection, just as the syphilitic fetus or infant succumbs, its body tissues literally alive with parasites. With this mechanism the untreated syphilitic spontaneously gets the infection in hand, destroys an innumerable number of treponemata and localizes the remainder in a fashion that is compatible with months and years of life.

Moore and Keidel<sup>2</sup> have recently pointed out that poorly-planned or inadequate treatment robs the patient with early syphilis of this immunity. If the natural course of the infection is interrupted by the administration of small, infrequent, sub-curative doses of treponemicidal drugs this natural provision for destroying and localizing the infection is thwarted, and this in an individual who still harbors parasites. It seems inevitable

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#### LEUKOCYTIC PICTURE

Infection of the throat may give four changes from normal in the leukocytic picture

- 1 Polymorphonuclear increase
- 2 Mononuclear increase
- 3 Leukopenia and diminution in, or absence of, granulocytes
- 4 Leukocytosis and relative hypogranulocytosis

Employing a broad definition for the word septic, all these four reactions may be the result of septic infection of the throat and mouth. That much is known, but the type of the infection is not known nor can the response to certain infections about which we do know something be predicated by the observed local manifestations

**Polymorphonuclear Increase**—In the first type of response, which may be exemplified grossly with an out-and-out pathogenic infection such as quinsy, there is a hyperleukocytosis and a hypergranulocytosis. Diphtheria also gives somewhat the same reaction, but not to the same extent. In a series of cases that were recently studied by Sowell<sup>1</sup> for the purpose of determining the effect of antitoxin on the leukocyte count, it was found that the total number of leukocytes in 33 cases was over 12,000 in 27 cases and under 11,000 in 6 cases. In the greater number of cases the count was over 20,000 with a pronounced increase in the number of polymorphonuclear cells. Of the 6 cases that were low, 2 of them presented a low polymorphonuclear count, both total and absolute. It is quite possible that these cases had a mixed infection and a type of response which might come under classification 3. That is to say, the ubiquitous diphtheroid, present in the mouths of so many individuals, ob-

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**Leukopenia and Hypogranulocytosis**—The third reaction occurs most beautifully in an interesting disease known as agranulocytic angina. In this condition, according to Schulz (*loc cit*) we have a syndrome of absence of granulocytic cells in the peripheral blood, jaundice, and a severe stomatitis, occurring for the most part in middle-aged females in whom there was absence of hemorrhagic diathesis, septic foci, and leukemia. These cases terminate fatally. In a few of the cases pyogenic organisms have been isolated and almost invariably have been found Vincent's organisms and spirochetes. In this same category belongs a disease to which on the whole comparatively little attention is paid from the point of view of hematology, that is, Vincent's angina. It is hard to get any definite information as to an accurate series of blood-counts made on patients with Vincent's angina. Simon, for example, says that the leukocyte count averages around 10,000. Weiss says that usually there is a decline in the polymorphonuclears and simultaneously an increase in monocytes and lymphocytes when the deposit is of a membranous, necrotic character in which the examination of the contents shows the presence of typical spirilla and fusiform bacteria. In this condition there is often a reduction in the total number of leukocytes, though this does not necessarily follow.

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**Case II**—A month before the admission of this boy to the ward, his brother, A H, was admitted with what was supposed to be streptococcic sore throat of some days' duration This little colored boy was four years old Eleven days before he was admitted he became sick with high fever and a rash Two days later a membrane appeared on the tonsils Despite diphtheria antitoxin his temperature continued and the throat failed to clear up When he was admitted to the contagious disease service he was found to have a dirty-appearing, widespread ulceration of the pharynx His total count upon admission was 19,400 white cells, which gradually fell in the course of ten days to 12,000 and the polymorphonuclears, which were 80 per cent fell to 68 per cent Improvement of the condition continued and the patient was discharged apparently in perfect condition

These two children evidently had scarlet fever and showed the leukocytic response that goes with this condition of streptococcic infection of the throat, whether due to a specific or a non-specific type of organism Innumerable other cases of leukocytosis with polymorphonuclear increase could be recounted Such a blood-picture is so common in peritonsillar or retropharyngeal abscess and diphtheria that it hardly seems worth while to go into such case histories

## II THE RESPONSE TO THROAT INFECTION CHARACTERIZED BY THE FORMATION OF AN EXCESSIVE NUMBER OF MONONUCLEAR CELLS

**Case III**—A B, a pupil nurse in one of the Philadelphia hospitals, was seen by me at the request of a nose and throat specialist, who thought that it would be advisable for an internist to see her in order to take care of her general condition She had been sick several days with a decidedly sore throat which on examination was found to be diffusely reddened and slightly edematous but without any evidence of ulceration The glands in the neck were palpable, as were the axillary glands Throat smears had been negative for diphtheria and for the organisms of Vincent's angina A blood count showed that the total leukocytes were 13,600, of which 74 per cent were classified as lymphocytes The blood count was repeated every day and gradually, with the subsidence of fever and the disappearance of the anginal symptoms, the leukocytic picture returned to normal

In this particular instance we have a patient who has one of the types of throat reactions in which there is marked increase in the number of mononuclear cells and, more particularly, of the monocytes The cells were classified as small lymphocytes and on more careful study it was found that there were a considerable number of them—true monocytes rather than the so-called small lymphocytes

numbered 12,500 per cu mm Polymorphonuclears were 89 per cent The throat cultures were all negative for diphtheria

**Case II**—A month before the admission of this boy to the ward, his brother, A H, was admitted with what was supposed to be streptococcic sore throat of some days' duration This little colored boy was four years old Eleven days before he was admitted he became sick with high fever and a rash Two days later a membrane appeared on the tonsils Despite diphtheria antitoxin his temperature continued and the throat failed to clear up When he was admitted to the contagious disease service he was found to have a dirty-appearing, widespread ulceration of the pharynx. His total count upon admission was 19,400 white cells, which gradually fell in the course of ten days to 12,000 and the polymorphonuclears, which were 80 per cent fell to 68 per cent Improvement of the condition continued and the patient was discharged apparently in perfect condition

These two children evidently had scarlet fever and showed the leukocytic response that goes with this condition of streptococcic infection of the throat, whether due to a specific or a non-specific type of organism Innumerable other cases of leukocytosis with polymorphonuclear increase could be recounted Such a blood-picture is so common in peritonsillar or retropharyngeal abscess and diphtheria that it hardly seems worth while to go into such case histories

## II THE RESPONSE TO THROAT INFECTION CHARACTERIZED BY THE FORMATION OF AN EXCESSIVE NUMBER OF MONONUCLEAR CELLS

**Case III**—A B, a pupil nurse in one of the Philadelphia hospitals, was seen by me at the request of a nose and throat specialist, who thought that it would be advisable for an internist to see her in order to take care of her general condition She had been sick several days with a decidedly sore throat which on examination was found to be diffusely reddened and slightly edematous but without any evidence of ulceration The glands in the neck were palpable, as were the axillary glands Throat smears had been negative for diphtheria and for the organisms of Vincent's angina A blood count showed that the total leukocytes were 13,600, of which 74 per cent were classified as lymphocytes The blood count was repeated every day and gradually, with the subsidence of fever and the disappearance of the angular symptoms, the leukocytic picture returned to normal

In this particular instance we have a patient who has one of the types of throat reactions in which there is marked increase in the number of mononuclear cells and, more particularly, of the monocytes The cells were classified as small lymphocytes and on more careful study it was found that there were a considerable number of them—true monocytes rather than the so-called small lymphocytes

tion to explain the fever and because of the blood count, despite the higher percentage of granulocytes which were, however, absolutely decreased, the patient was thought to have aplastic anemia and was so diagnosed. The mouth at no time had the frightfully nasty necrotic ulcerative lesions that occur in agranulocytic angina.

#### CASES OF VINCENT'S ANGINA

**Case VI—C L**, a young negress of twenty seven years, was admitted to the hospital on account of sore mouth, pains in the head, and weakness. She had the sore mouth for several weeks prior to admission. The explanation of the sore mouth was revealed when the buccal cavity was examined. There was marked salivation. The tongue was red and beefy and along the edges and tip of the tongue were ulcers with a necrotic membrane. The surface of both cheeks was also covered with this necrotic, ulcerative membrane. Beneath the tongue the same condition was present. This patient on admission showed a total red count of 4,250,000, with a hemoglobin of 80 per cent. The white blood cells numbered 4250 of which 50 per cent were polymorphonuclears. In this case, then, the white count showed a leukopenia and a polymorphonuclear decrease which was both absolute and relative. The smears from the mouth showed many spirochetes and fusiform bacilli.

**Case VII—N P**, a young colored woman aged twenty years admitted to the hospital on account of sore throat and sore mouth, which she had had for five days. Examination of this young woman's mouth showed the entire buccal membrane reddened and congested. There was a necrotic ulcerative area on the cheek surface near the angle of the jaw and more anteriorly a smaller patch. The tonsils were markedly enlarged and covered with a firmly adhesive membrane. As in the previous case, the examination, except for the local condition, was entirely negative. In neither case were positive cultures gotten for *Bacillus diphtheriae*. The smears showed many fusiform bacilli and spirochetes. This case showed a low white count. Three days after admission to the hospital her count, which upon admission had been 4500 per cu mm, had fallen to 4000. Of these 4000 cells, 50 per cent only were polymorphonuclears, showing both an absolute and relative polymorphonuclear decrease. The mouth condition improved rapidly and the young woman was discharged from the hospital one week after admission.

**Case VIII—E J** was a third case of Vincent's angina in a young colored woman, who was in the hospital for eleven days, gaining admission on account of difficulty in swallowing and swelling of her neck. These acute symptoms she had only noticed for a day and the explanation lay in the greatly congested mucous membrane which showed four or five ulcerated and necrotic areas. Cultures from these areas showed many fusiform bacilli and spirochetes but no diphtheria organisms. Under vigorous treatment the mouth condition improved rapidly. The patient was discharged in apparently excellent condition. This patient also showed a leukopenia, the first count showing 4500 cells and the second count four days later showed 3750 white cells. The differential figures for the first count showed 45 per cent of small mononuclears, 42 per cent of polymorphonuclears, and on the second showed 54 per cent

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ination of this boy's mouth, the whole interior seemed to be gangrenous and sloughing. Cultures from the mouth showed the presence of numerous fusiform bacilli and spirochetes. There was some slight degree of adenopathy but with only a few small, hard, and shot-like, superficial glands, palpable. The blood count in this boy showed the total leukocytes on several examinations to be over 100,000, with the cells of the granulocytic group varying between 2 and 3 per cent. In spite of all treatment the boy rapidly went on to death.

Last year Dr. George Patrick Quinn reported before the student body of Tulane a patient with oral sepsis who had the blood and mouth picture of leukemia. In this particular history the patient had thirteen teeth removed and following this developed a mouth infection. When he was admitted to the hospital it was found that the gums were swollen, spongy, and presented a widespread necrosis and gangrene of the mucous membrane involving the buccal cavity, with the characteristic very foul odor of mouth infections. The patient was found to have marked leukocytosis, 408,000 cells being present in the peripheral blood. Two days later they had risen to 419,000, with 93 per cent lymphocytes. The cells were classified as an immature form of lymphocytes, lymphoblasts, which was confirmed by the absence of granules in the ectoplasm of the cell when stained by the oxidase method. The patient died the day following this blood-count. In this particular instance they were apparently dealing entirely with a case of acute leukemia of the so-called lymphatic type. I know of no other condition in which there is such an enormous increase of white cells, a fact which would rule out cases with a leukemoid blood-picture, but due to some other cause.

An interesting thing in these 2 cases of leukemia mentioned above is that the acute fulminating symptoms developed concomitant with the mouth infection. In both these instances apparently the condition of the patient was excellent until the mouth infection occurred. It is probably reasonably fair to assume, though this is not by any means definite, that an acute lymphatic leukemia was present prior to the mouth infection, but when this took place very promptly the more spectacular acute symptoms developed.

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One of us (H M B) in studying various members of a very large family has discovered 18 individuals in four generations with glycosuria, one has a mild symptomless diabetes mellitus, the others have an apparently benign glycosuria, some excreting sugar constantly, others intermittently. What is it, therefore, in a given family which determines whether an individual develops a diabetic or a non-diabetic glycosuria? We hope that future investigators will solve this problem.

It has only been within the past decade that the various glycosurias have been separated into different groups. This has been accomplished by utilizing a combined and simultaneous series of observations on patients after the ingestion of sufficient glucose to produce definite changes in the blood-sugar, respiratory quotient, and urinary sugar. As a result of numerous observations made by many investigators, we are permitted to formulate certain ideas concerning the mechanism of the glycosurias, which may be grouped as follows:

A Patients with glycosuria having a normal or subnormal fasting blood-sugar level, with normal utilization of glucose

1 Renal glycosuria

2 Cyclic renal glycosuria, or alimentary glycosuria

B Patients with glycosuria, having a normal fasting blood-sugar level, but with an inability to readily mobilize and store increased doses of glucose

Diabetes insipidus

C Patients with glycosuria having a normal or an increased fasting blood-sugar level and an inability to normally utilize, mobilize, and store glucose

Diabetes mellitus

Practically all patients who have sugar in their urine may be placed in one of these three groups. A discussion of some of the factors involved in the production of these various types of glycosuria will be given as each condition is brought to your attention.

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TABLE 1

Date		Loc		Time		Wind		Temp		Humidity		Pressure		Clouds		Remarks	
Day	Month	Year	Lat	Long	Hour	Min	Sec	Dir	Speed	Air	Sea	Wet	Dry	Bar	Wind	Sea	Notes
10	10	1968	10° 34'	156° 00'	06	00	00	10	10	28.0	10	75	75	1010.0	10	10	10
11	10	1968	10° 34'	156° 00'	07	00	00	10	10	28.0	10	75	75	1010.0	10	10	10
12	10	1968	10° 34'	156° 00'	08	00	00	10	10	28.0	10	75	75	1010.0	10	10	10
13	10	1968	10° 34'	156° 00'	09	00	00	10	10	28.0	10	75	75	1010.0	10	10	10
14	10	1968	10° 34'	156° 00'	10	00	00	10	10	28.0	10	75	75	1010.0	10	10	10
15	10	1968	10° 34'	156° 00'	11	00	00	10	10	28.0	10	75	75	1010.0	10	10	10
16	10	1968	10° 34'	156° 00'	12	00	00	10	10	28.0	10	75	75	1010.0	10	10	10
17	10	1968	10° 34'	156° 00'	13	00	00	10	10	28.0	10	75	75	1010.0	10	10	10
18	10	1968	10° 34'	156° 00'	14	00	00	10	10	28.0	10	75	75	1010.0	10	10	10
19	10	1968	10° 34'	156° 00'	15	00	00	10	10	28.0	10	75	75	1010.0	10	10	10
20	10	1968	10° 34'	156° 00'	16	00	00	10	10	28.0	10	75	75	1010.0	10	10	10
21	10	1968	10° 34'	156° 00'	17	00	00	10	10	28.0	10	75	75	1010.0	10	10	10
22	10	1968	10° 34'	156° 00'	18	00	00	10	10	28.0	10	75	75	1010.0	10	10	10
23	10	1968	10° 34'	156° 00'	19	00	00	10	10	28.0	10	75	75	1010.0	10	10	10
24	10	1968	10° 34'	156° 00'	20	00	00	10	10	28.0	10	75	75	1010.0	10	10	10
25	10	1968	10° 34'	156° 00'	21	00	00	10	10	28.0	10	75	75	1010.0	10	10	10
26	10	1968	10° 34'	156° 00'	22	00	00	10	10	28.0	10	75	75	1010.0	10	10	10
27	10	1968	10° 34'	156° 00'	23	00	00	10	10	28.0	10	75	75	1010.0	10	10	10
28	10	1968	10° 34'	156° 00'	24	00	00	10	10	28.0	10	75	75	1010.0	10	10	10
29	10	1968	10° 34'	156° 00'	25	00	00	10	10	28.0	10	75	75	1010.0	10	10	10
30	10	1968	10° 34'	156° 00'	26	00	00	10	10	28.0	10	75	75	1010.0	10	10	10
31	10	1968	10° 34'	156° 00'	27	00	00	10	10	28.0	10	75	75	1010.0	10	10	10
1	11	1968	10° 34'	156° 00'	28	00	00	10	10	28.0	10	75	75	1010.0	10	10	10

(f) With no dietary control—after one month of restricted diet—the patient, during a period of three years, has not had any symptoms of diabetes. Neither has there been any evidence of acidosis.

(g) It is possible that the constant loss of glucose in the urine might have influenced the retarded physical development of this patient

(h) Because of the age of the patient it was impossible to make metabolic studies

The evidence presented classifies this patient as belonging to a well-recognized group of individuals who constantly excrete sugar in the urine and have a fasting blood-sugar level which is either normal or subnormal. During a glucose tolerance test, in which 50 or 100 gm of glucose dissolved in 300 cc of water with the juice of one lemon is given on a fasting stomach—the majority of these patients respond with a practically normal blood-sugar curve, and a varying glycosuria, the extent of

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before returning to the normal level. Some evidence has been presented by Holst,<sup>3</sup> Faber,<sup>4</sup> Malmros,<sup>5</sup> and others that cases of this type differ from mild diabetes. The points of differentiation are not clear, but when these patients are observed over a long period of time they do not develop symptoms of diabetes. Stress is laid upon the fact that most of these patients have a normal fasting blood-sugar, this is of great importance in differentiating between the benign type of glycosuria and mild diabetes. At present we are justified in admitting the existence of this condition, but patients so classified should be closely observed over a period of years. If a group of such patients could be studied during the progress of an acute infection valuable information could be obtained concerning the behavior of the blood and urinary sugars. As a result of the altered metabolism one could determine the similarity or non-similarity to diabetic behavior.

**3 Diabetes Innocens**—This term is applied to an unusual clinical condition which shows certain characteristics of diabetes mellitus, but apparently runs a harmless course. Such cases have been described by Salomon,<sup>6</sup> Frank,<sup>7</sup> and Riesman.<sup>8</sup> Not all of their cases are easily classified since no detailed observations were made on the blood-sugar after the ingestion of glucose or food.

Campbell<sup>9</sup> emphasizes the following points in the differentiation of diabetes innocens from diabetes mellitus. The discovery of glycosuria is accidental. There are no symptoms of diabetes except glycosuria, and such symptoms do not occur despite the long duration of the condition and absence of dietary regulation. Sugar is present in the urine with normal fasting and postprandial blood-sugar levels. There is an increase in glycosuria with increased carbohydrate intake, but the increase is not commensurate with the increase in food. Respiration studies show high respiratory quotients, demonstrating carbohydrate utilization and other features incompatible with the diagnosis of mild diabetes.

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and rarely a hyaline cast. The urine was dextrorotary and fermented with yeast. Crystals were formed with phenylhydrazin. Seliwanoff's test for levulose was negative. The hemoglobin, red blood-cell and white blood cell counts, and differential white-cell count were within normal limits. The blood Wassermann was negative. The fasting blood sugar was 114 mg per 100 c c of blood.

The patient was placed on a diet of carbohydrates, 80 grams, protein, 80 grams, and fat, 180 grams accompanied by insulin, 15 units in the morning and 10 units in the evening. Tests of separate voidings carried out for thirty-six hours showed a sugar-free urine in only one specimen on this diet and insulin dosage. (He had remained in the hospital several days on this regime without becoming sugar-free when he found it necessary to return home.)

He returned for further study July 19, 1927, stating that he had adhered to the above diet and insulin dosage for seven weeks without becoming sugar-free or gaining in strength. His naked weight was 129½ pounds. His fasting blood-sugar was 102 mg per 100 c c of blood. On a diet of carbohydrate, 75 grams, protein 80 grams, fat 170 grams, and 25 units of insulin daily for a period of two days, his average glucose excretion was 11.5 grams or 8.3 per cent of the available glucose in the diet. While receiving this diet and insulin dosage, nine single specimens of urine were tested during twenty-four hours, and all of these showed sugar.

On July 29th 100 grams of glucose were given by mouth as lemonade, with the following results:

7-20-27	Blood-sugar Per cent	Urine sugar Per cent	Urine-sugar Grams
Fasting	0.112	0.7	1.75
Glucose, 100 grams by mouth			
20 minutes later	0.190		
40 minutes later	0.230	2.8	2.52
1 hour later	0.290	2.0	2.3
2 hours later	0.175	1.7	7.6

On August 2nd before breakfast his blood sugar level was 101 mg. An indwelling urethral catheter had been fastened in the bladder and 222 c c of urine containing 0.7 per cent of sugar had been removed at the time of collection of the blood specimen. Forty units of insulin was injected subcutaneously. The bladder was irrigated at intervals with sterile saline solution. Water was taken in large quantities by mouth. The bladder was emptied every two or three minutes, and the separate specimens of urine were tested with Benedict's solution. In one hour and twenty-five minutes the bladder-urine became sugar-free for the first time, the simultaneous blood sugar level was 73 mg. There were no symptoms of an hypoglycemic reaction. Within one minute the patient took 100 grams of glucose by mouth as lemonade and the testing of urine specimens was continued. Sixteen minutes after the ingestion of glucose Benedict's solution again showed definite presence of sugar in the urine. The simultaneous blood sugar level was 120 mg.

August 3rd simultaneous blood and urine specimens were collected one

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(c) The urine always contained sugar but no acid bodies

(d) While receiving weighed diets the amount of glucose excreted in the

TABLE 2

Date 1928	Ibkd weight pounds	D I E T					U R I N E			Blood Sugar Per Cent fasting	S U M M A R Y			Response to graded diets = increased g carbohydrate content and approximately isocaloric value
		Carbohydrate Grams	Protein Grams	Fat Grams	Calori- es	Available Glucose Grams	Glucose in Urine		Total available Glucose each diet period Grams		Total Glucose ex- cretion each diet period Grams	Percent total avail- able Glucose excreted each diet period		
							Reduction Per Cent	Total Grams						
8-14		100	63 0	150	2002	151 5	2575	0 95	4 5	0 070				
8-15		100	63 0	150	2000	151 5	3005	0 76	2 7					
8-16		91	61 5	150	1960	141 7	1465	1 85	27 1					
8-17		100	63 0	150	2000	151 5	36 0	1 00	36 2		596 2	110 5	18 5	
8-18		160	61 5	116	1930	207 3	4075	0 79	3 3					
8-19 147		160	61 5	116	1930	207 3	3335	0 84	28 0					
8-20 147 1/2		140	61 5	116	1850	187 3	2950	1 18	34 7		601 9	95 0	15 8	
8-21 148 1/4		230	60 5	90	1972	274 0	3 5	1 00	23 2					
8-22 148 1/4		230	60 5	90	1972	274 0	40	1 33	39 2					
8-23 150 1/4		220	60 5	90	1940	265 2	28 5	1 45	41 4		613 2	103 8	1 8	
8-24 150		300	61 5	67	2049	342 3	3800	1 11	42 2					
8-25 149 3/4		300	61 5	67	2049	34 3	3345	1 20	40 4					
8-26 150		300	61 5	67	2049	34 3	3000	1 35	39 5	0 085	1026 9	12 1	11 9	

urine was only slightly increased by raising the carbohydrate intake of the food

(e) With increases in the available glucose intake there was a decrease in the percentage of the available glucose excreted

(f) Glucose tolerance curves show an hyperglycemia at the end of one hour, such as is observed in diabetics, the hyperglycemia is more prolonged than a normal response to the ingestion of glucose

(g) Spontaneous hypoglycemia occurred three hours after the last glucose tolerance test which we assume is very good evidence that the pancreas is able to supply a good quality and quantity of insulin

(h) The renal threshold in this patient is quite low. During the period of observation his urine was never sugar-free, even with a low blood sugar level, or when insulin was given in an effort to promote better carbohydrate utilization. The renal threshold, even when roughly tested by the method we have described, resembles in all respects that of renal glycosuria. The variation in the blood sugar level at which the urine ceased to show sugar and again contained sugar is in keeping with observations by others in this condition.

4 Diabetes Mellitus—It is unnecessary to say much concerning this condition. Everyone is familiar with the many and extensive studies which have been made on patients with diabetes mellitus and on animals with partial and complete pancreatectomies. In the diabetic patient there is a disturbance of

- (c) The urine always contained sugar but no acid bodies  
 (d) While receiving weighed diets the amount of glucose excreted in the

TABLE 2

Date 1928	Ibkd weight pounds	DIET					URINE			Blood Sugar Per Cent fasting	SUMMARY			Response to graded diets & increased g carbohydrate content and approximately isocaloric value
		Carbohydrate Grams	Protein Grams	Fat Grams	Calori- es	Available Glucose Grams	Glucose in Urine		Total available Glucose each diet period Grams		Total Glucose ex- cretion each diet period Grams	Percent total avail- able Glucose excreted each diet period		
							Reduction Per Cent	Total Grams						
8-14		100	63.0	150	2002	151.5	2575	0.95	4.5	0.070				
8-15		100	63.0	150	2000	151.5	3005	0.76	2.7					
8-16		91	61.5	150	1960	141.7	1465	1.85	27.1					
8-17		100	63.0	150	2000	151.5	3000	1.00	36.2		596.2	110.5	18.5	
8-18		160	61.5	116	1930	207.3	4075	0.79	3.3					
8-19	147	160	61.5	116	1930	207.3	3335	0.84	28.0					
8-20	147 1/2	140	61.5	116	1840	187.3	2940	1.18	34.7		601.9	95.0	15.6	
8-21	148 1/4	230	60.5	90	1972	274.0	3000	1.00	23.2					
8-22	148 1/4	230	60.5	90	1972	274.0	4000	1.33	39.2					
8-23	150 1/4	220	61.5	90	1940	265.2	2800	1.45	41.4		615.2	103.8	1.6	
8-24	150	300	61.5	67	2049	342.3	3800	1.11	42.2					
8-5	149 3/4	300	61.5	67	2049	342.3	3345	1.20	40.4					
8-26	150	300	61.5	67	2049	342.3	3000	1.35	39.5	0.085	1026.9	12.1	11.9	

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Sanatorium and hygienic-dietetic treatment of tuberculosis is efficient and sound, but to many individuals it is out of the question because of lack of money. It is a sad fact that a multitude of the tuberculous "cannot afford to get well." Recovery from definite pulmonary tuberculosis, by which is meant definite physical signs, definite x-ray findings, and definite symptoms, cannot possibly occur under one year, and in the vast majority of open cases, this time is doubled and frequently trebled or quadrupled. The man or woman of moderate means, face to face with an economically unproductive period of one to four years, runs up against an "impasse." The patient, as above stated, "cannot afford to get well."

Collapse therapy, in addition to being a potent factor in diminishing symptoms, in lessening poison absorption, and in bettering the clinical condition of the patient, is a great "short-cut" to recovery. Anything that reduces the time necessary to recovery from pulmonary tuberculosis is of inestimable advantage to the patient, for it is the attrition of the disease rather than the disease itself, that wears down many.

The basis of all collapse therapy is of course rest to the diseased organ. Rest is looked upon as the cornerstone of treatment in tuberculosis, yet the damaged lung moves from eighteen to twenty-four times per minute in its respiratory excursions. The whole problem of collapse therapy rests upon lung immobilization.

What, then, are the indications? They may be very briefly stated:

- 1 Unilateral active disease especially if there is evidence of cavity formation

- 2 Bilateral disease where by physical and x-ray examinations there is no activity in the process of the less involved lung

Cases presenting equal or approximately equal bilateral involvement are unsuited for collapse therapy. Certain selected cases can be subjected to modified collapse therapy on both sides, but these cases are wholly within the domain of the expert and have no place in this discussion. There are certain contra-

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a mine which may at any moment explode and bury him beneath the débris of exsanguination, general dissemination of the disease process, or tuberculous pneumonia

Thus forewarned, what procedure comes naturally to the mind of the attending physician? Artificial pneumothorax In those cases in which it is successfully and completely induced the result is such as to constitute veritably one of the miracles of modern medicine There is no more dramatic change in the clinical condition of an acutely ill tuberculous individual than that brought about by a complete unilateral lung compression The abrupt fall in temperature, the lessening and disappearance of cough and sputum, the improvement in the general sense of well-being, the gain in weight and strength all these arriving concurrently, seem to constitute a veritable magic of therapy

But this is the ideal case! And in too many this ideal is not realized To be sure, in the large majority of those in whom a workable artificial pneumothorax is obtained there is seen marked improvement *up to a certain point* and at that point, the patient remains stationary Cough and sputum have been appreciably reduced Fever has disappeared Weight has been gained, and therewith strength The patient is clinically better, but the patient is not clinically well, and as time passes by both patient and physician become weary of the incessant "marking time" A life may have been saved, but an economic recovery is far from being achieved

Why is this? It is because a partial but not a total pulmonary compression has been obtained Various areas of parenchymal involvement have ceased to function as active foci, but the seat of the trouble, the cavity, or cavities, have, because of the presence of parietal pleural adhesions, proved incapable of compression Fairly high air-pressures may be tried in the hope (usually vain) of breaking an adhesion Very high pressures are risky because of the fact that if an adhesion should give way there is no guaranty on which end it will yield, and, should it be on the pulmonary end, tearing of lung tissue with resultant spontaneous pneumothorax, infection, pyopneumothorax, and all its melancholy train of

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paratively simple one for the surgeon with an accurate knowledge of the triangles of the neck. It must be borne in mind, however, that there are several anatomic aberrations possible on the part of the phrenic nerve, and the surgeon about to undertake this procedure should familiarize himself with them in order not to be dismayed if, upon exposure of the anterior surface of the scalenus anticus muscle the nerve is not at once plainly seen.

Still another method can be employed for converting partial into total compression. Professor Jacobaeus, of Sweden, in 1913, devised a method of severing string or band-like adhesions that acted as guy-ropes to keep cavities from collapsing. By means of an electrically-lighted tube analogous to a cystoscope, and called a thoracoscope, he directly investigated these adhesions and, when deemed suitable, he severed them by introducing through a separate opening in the pleural cavity an electrocautery by means of which, when heated to a dull-red, he slowly seared the adhesion until it gave way and the wall of the cavity was no longer subject to traction and could, therefore, collapse. It was found that adhesions thus directly explored frequently presented a very different appearance from that viewed in the  $\gamma$ -ray film, for while in the latter they could be seen but in one plane, upon investigation with the thoracoscope linear appearing adhesions were found to be broad and band-like and other differences not demonstrated roentgenologically were apparent, while, in addition, many adhesions were seen which had been invisible on the  $\gamma$ -ray film. Cases, therefore, which from the  $x$ -ray picture appear wholly suitable for this procedure, upon thoracoscopic examination may be found impracticable for its application. Thick band-like adhesions above the third rib are almost uniformly unsuitable for cauterization. The method is not without its dangers. Cauterization must be done slowly and with the cautery at only a dull-red heat in order to sear the blood-vessels that will be encountered in the adhesions. Bright red or white heat will too rapidly burn through the vessels and will predispose to hemorrhage. It is of paramount importance to let the cautery under no circumstances penetrate the wall of a cavity, as then a spontaneous pneumothorax is brought about with its train of infection, tuberculous empyema,

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three, four, or even five stages, as postoperative shock is thus lessened

The actual operative mortality of this procedure in the hands of skilled men, is not great and the results are on the whole satisfactory. A complete thoracoplasty is by no means always necessary. With an apical lesion a partial thoracoplasty can be done, involving the number of ribs that must be sacrificed in order to obliterate the lesion. When preceded by phrenico-exeresis, frequently the partial compression so obtained is sufficient to reduce the extent of major surgical intervention.

To recapitulate then, the ideal form of collapse therapy is artificial pneumothorax. Simple to induce, practically painless, almost wholly free of danger and, when complete, the most productive of clinical results, it stands today an enduring monument to its originator, Carlo Forlanini of Pavia.

If artificial pneumothorax is found to be wholly inapplicable, then of course the Jacobaeus method of adhesion-cauterization is out of the question, as it can only be employed when a partial pneumothorax exists. The two operations of phrenico-exeresis and of extrapleural thoracoplasty remain. In cases with a definitely thickened pleura over the lower lobe of the affected side, with evidences of thick strong pleural adhesions, it is very doubtful whether phrenico-exeresis will produce sufficient rise in the dome of the diaphragm to warrant its performance. In such cases it is best to proceed at once with the thoracoplasty. It has been said that a good pneumothorax is better than a thoracoplasty, but that a good thoracoplasty is better than a bad pneumothorax (Claus).

The cases requiring nicety of judgment are those in which a partial pneumothorax has been obtainable. No two cases are alike, and each must be judged on its own merits. The surgeon that is interested in collapse therapy must have a good knowledge of pulmonary tuberculosis, and the physician that is interested in pulmonary tuberculosis must have a general acquaintance with the various methods of surgical intervention, what they seek to attain, how they seek to attain it, what risk is involved, what limitations present themselves, and what in-

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So let us weigh everything carefully We have today methods at our command which are invaluable in aiding a diseased lung upon its way to cicatrization We have the same age-old natural processes that have effected so many recoveries throughout the centuries Both elements must be kept in mind, those under the control of man and those under the control of nature, and the method of procedure must be decided upon after a careful estimation of the value of each as expressed in the condition of the patient Too great enthusiasm will lead us on the rocks of meddlesome surgery, too great conservatism will make us responsible for the loss of valuable lives that might and should have been indefinitely prolonged Ours is the choice for better or for worse Hippocrates summed it all up in his famous aphorism

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Fig 222 —Lateral view showing the ham-like right hand and swollen lower half of right forearm (November 1, 1928)



Fig 223 —Hand after treatment on discharge of patient from hospital (December 18, 1928)

right wrist followed in a week by swelling of the entire hand and forearm. There was much pain and tenderness, particularly on use. From June to November, 1928 there was no improvement in spite of all treatment and she complained not only of pain in the wrist and hand but of nervousness and insomnia.

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Her previous diseases appear interesting from their possible bearing on her nervous system. She had measles and whooping-cough complicated by pneumonia at six, pneumonia again at eight, and scarlet fever at ten. At thirteen she complained of back pain but no pathology was revealed. At this time she had a menorrhagia but after four months appeared well. Menstruation began at twelve and has always been irregular. The periods last five days and come every three weeks, occasionally every ten days, and occasionally every thirty days. Three months ago the menstruation lasted two weeks. There has never been any pelvic pain. The sensitiveness to pain is evident.

Physical examination revealed a perfectly normal girl of sixteen with the exception of a swollen right upper extremity. She had large eyes, placid face, rather extremely sensitive nature, easily moved to tears and smiles. The complexion was smooth and clear, eyes blue, hair blonde, and she impressed one as being emotionally unstable and of a decidedly neuropathic constitution.

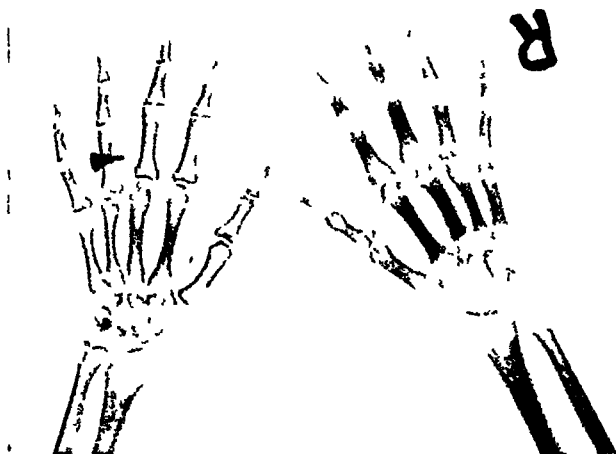


Fig. 226—Same as Fig. 224, except taken on December 20, 1928

tion. She had complained so much about the pain in the arm that the fore arm and hand had been bandaged and supported by a splint. She could move the fingers only slightly because of the tenseness of the skin and the marked edema. It was a spade-like hand, the greatest edema being over the dorsum, and pitting was easy. It had the same appearance as a foot with the huge edema of congestive heart-failure.

Laboratory examinations were negative throughout, including urine, blood, gastric contents, stool, and basal metabolism. The teeth and sinuses were normal, though the x ray showed the lower ends of the radius and ulna, the metacarpals and the ends of all the phalanges of the right upper extremity to possess a decided translucency, apparently a definite decrease in their calcium content. The lateral view showed this more definitely than the anteroposterior plate. The hip girdle and bones of the lower extremities were normal.

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- 1 Angioneurotic edema
- 2 Milroy's hereditary edema, which apparently does not appear above Poupart's ligament
- 3 Constant edema with periodic exacerbations, of neuropathic origin
- 4 Intermittent articular dropsy, which is not to be confused with Charcot's joint of locomotor ataxia. It may come on periodically and the skin over the joint is neither hot nor red. It seems a part of a general neurosis

However, such cases do not occur except relatively rarely and no one clinician has had very much experience with the group as a whole. Cases of angioneurotic edema and Milroy's hereditary edema are easy of diagnosis and capable of classification. Cases that fall into divisions three and four are more susceptible to difference of opinion.

While in the beginning the complaint in this patient seemed to have begun over in the right wrist there was no evidence that there was any arthritis in this joint, and for months the mass of the edema was in the tissues about the metacarpal bones and the hyperesthesia in the skin and tips of the fingers of the right hand.

This case seems to belong to the third group of neuropathic edema with exacerbations and having a neuropathic origin. Neuropathic dropsy is more expressive of the condition than is neuropathic edema because the pitting was extreme and the drainage excessive. The edema gradually decreased. The middle and upper third of the forearm and the lower one-third of the arm did not pit. It is said that the children of an alcoholic parent tend more to angioneurotic edema. The father of this patient was apparently suffering with dipsomania and died of acute alcoholism. Mueller, of the Mayo Clinic, presented in November, 1928, a case of angioneurotic edema appearing in a seven-day cycle. Oppenheim, on page 1325 of his text-book of "Nervous Diseases," calls attention to the periodic occurrence of articular dropsy. Sydenham talked of edema in hysteria and Charcot tried to lay the foundation for a Charcot type of angioneurotic edema. Angioneurotic edema recurring at definite intervals and intermittent articular dropsy do occur. "Blue edema" is the

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Many physicians had been consulted over this five-year period. Each had made a diagnosis of cystitis because of the continued pus in the urine. And various other contradictory diagnoses were made. One doctor had

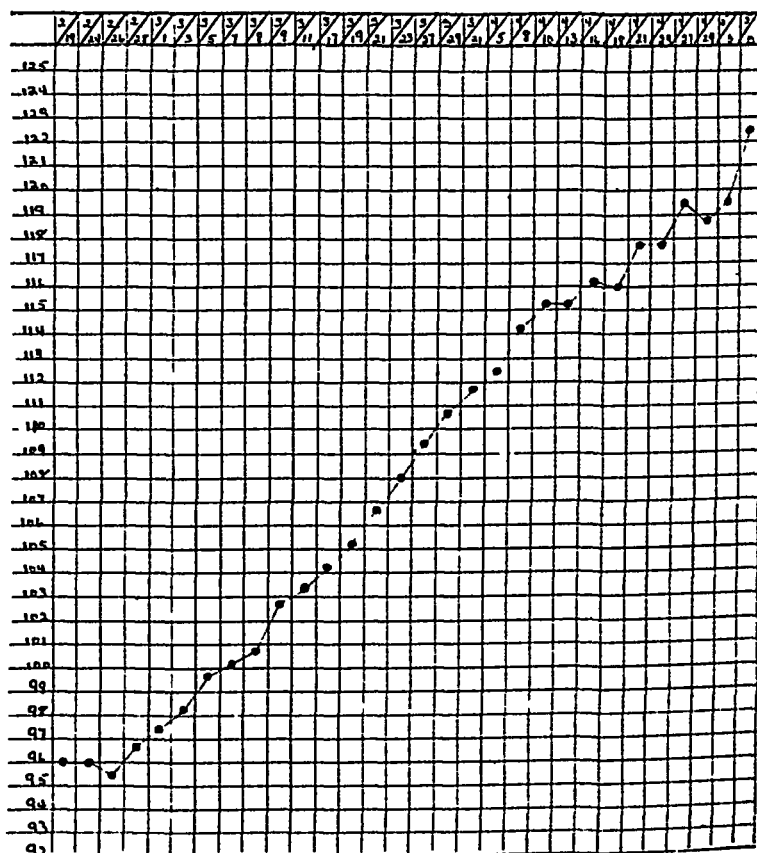


Fig 227 —Note weight on entrance, Feb 2, 1928 (96 pounds) and (122 pounds) on dismissal May 5, 1928

advised a pan-hysterectomy and another advised against it. One doctor had advised the removal of all the teeth and another had seen no reason for the removal of any of the teeth.

Some strange things had happened to her in the course of the last five years. She was formerly never troubled with gas or indigestion and now gas

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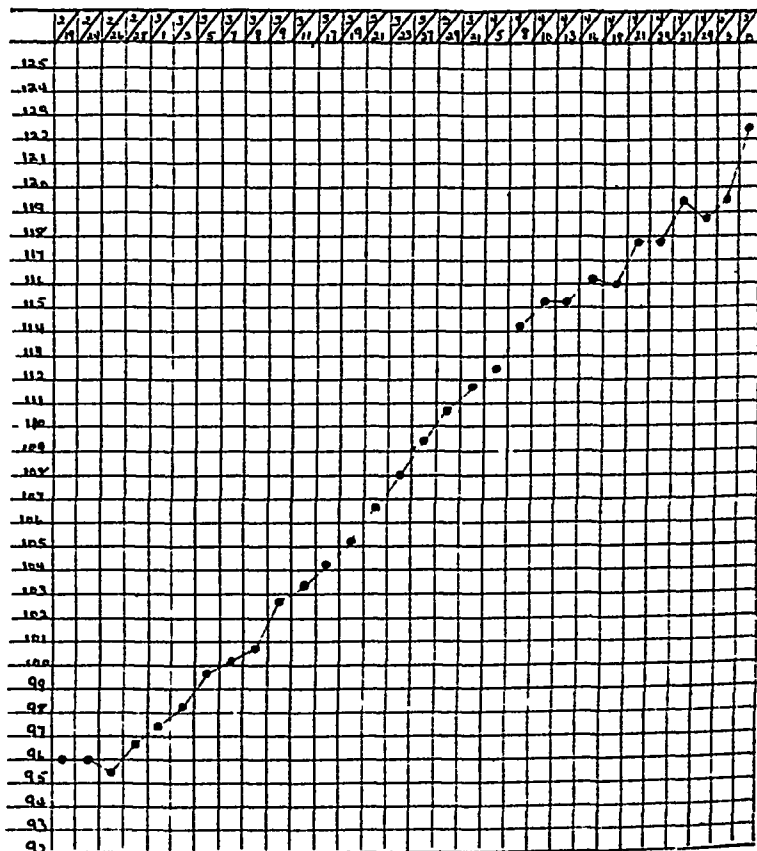


Fig 227 —Note weight on entrance, Feb 2, 1928 (96 pounds) and (122 pounds) on dismissal May 5, 1928

advised a pan-hysterectomy and another advised against it One doctor had advised the removal of all the teeth and another had seen no reason for the removal of any of the teeth

Some strange things had happened to her in the course of the last five years She was formerly never troubled with gas or indigestion and now gas



bility and a gradual but definitely apparent deterioration of the mind, all associated with an exaggeration of the deeper reflexes

She was put in the hospital on the same date of her arrival at the clinic, February 18th, and four days later, February 22d, she was disoriented both in time, place, and person, and was convinced she was doomed to die. Fortunately she could and did eat and she was given a 2500 calorie, high protein diet with emphasis on meats, milk, eggs, peas, and beans. Brewer's yeast in 2-dram doses was given after meals, sedatives forbidden, and dilute hydrochloric acid and tincture of nux vomica given in 1½ c c doses after meals. Her window was kept open constantly in the coldest winter weather, water and milk served ice cold, she was permitted to keep her hands and burning feet exposed to the cold air, the heat was turned off in her room and she was given a tepid tub bath at ten in the morning and four in the afternoon, daily. Ten days later, February 28th, the hydrochloric acid was increased to 2 c c, an occasional dose of castor oil was given on waking in the morning. On account of her psychosis two nurses were necessary from February 18th to March 2d, but on March 9th both special nurses were discharged, so marked was the improvement. There were lapses into disorientation but finally the patient was controlled mentally by the advice that "for the present everything she thought was wrong, and her duty to herself and to her husband was simply to eat what was brought her, and that time would bring about complete recovery." March 12th progress note of the hospital record testates "A gradual improvement is continually noted. Complaints of hands and feet. Says they feel tired, hands feel puffy and they burn. She knows now, since her improvement, how weak she is." Between March 2d and March 20th there were several attacks of acute labyrinthitis, a sea-wave bed, a typical Ménière's syndrome. She describes these attacks as if the bed is going to roll out from under her and most of the sensations passed from right to left. The burning sensation in hands and feet diminished gradually. Between February 18th and March 19th she had gained 9 pounds and weighed 105 pounds. On March 31st, a slight redness remained under the anterior third of the tongue but the mouth and hands were well. The feet "fired up" on the night of March 29th. Her weight was 111½ pounds. The diarrhea disappeared after the first ten days in the hospital. She was discharged from the hospital with a weight of 122½ pounds, or a total gain of 26½ pounds. During all this time there were occasional attacks of nervousness and of vague and transient pains. A few flurries of diarrhea occurred but she felt better by remaining in bed and eating freely. There was no fever at any time, the temperature usually ranging from 97° to 98° F, the pulse from 80 to 100 and as she improved and gained in weight she began to sleep normally, her temperature gradually rose to normal, pulse dropped to 80 and respiration to a level of 20. Her emotions gradually grew stable, her outlook cheerful and her mind normal. She was sent to the mountains of North Georgia for six months with the following directions (May 15, 1928)

- 1 For the next year be as happy, as natural, and as vacant minded as possible
- 2 Avoid any social, outside civil, or ecclesiastical duties, except purely as a matter of pleasure, and without strain

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- 1 For the next year be as happy, as natural, and as vacant minded as possible
- 2 Avoid any social, outside civil, or ecclesiastical duties, except purely as a matter of pleasure, and without strain

"However, I found myself slipping before this hot spell set in. My stomach and bowels run, with much gas in intestines, side and around heart, with throat and mouth sore, the bowels looser, one day four stools of pale yellow, spongy consistency, at which I left off yeast, frequency now diminished but texture the same, headache, shifting from front to rear of head, old feeling of swelling under left eye and right foot, at times over whole body, occasional little pimples on various parts of body, constant blisters and itching on toes, occasional bright spots in cheek, especially left side, some firing of limbs up to knees. The complexion, which has been clear is darker and cloudy looking with dark circles under eyes. And I do not sleep well at nights.

"When I was unable for several days to throw these troubles off, I got discouraged, and I am ashamed to confess, wrote you a blue letter and had a good cry. I did not let the folks here know it however, and now have myself in hand again and have torn up the letter. For I have been in fine spirits with good control and poise, and with judgment clear and reasonable. When Mr. — was here two weeks ago, he was delighted with condition and appearance.

"I have tried to follow instructions and have had fine co-operation and supervision on the part of father and mother and have succeeded admirably, except perhaps as to the injunction to "be vacant-minded." I can't do it. But I have surprised myself by the ease with which I am dismissing troublesome matters. Hope I'm not getting too selfish.

"Now, in describing these symptoms, I do not mean that they have returned with the same intensity as when I first went to the hospital but they are distressingly uncomfortable and have lasted longer than at any previous time since I began to get better.

"Do you think that vinegar or beets, or acid of fresh tomatoes, or any other special article of food would be apt to have bad effects? I have tried to study reasons for these recurrences but have reached no satisfactory conclusions.

"Have had one rather restricted menstrual period, accompanied by quite a bit of pain, but not as much as the former period which was the worst I had ever experienced. Another is about due but thought perhaps these cold sponges are delaying it. Would that have any bearing on all these other disturbances?

"Do you suggest that I increase the amount of exercising to try to control my weight? I find that I feel better on moderate amount of stirring around. I tried dusting and would fire up each time. I walk some but not a great deal."

Our reply was as follows:

"Thank you for your letter of July 5th. We had been expecting to hear from you.

"1. You are doing well. You must do a good bit of overlooking—more overlooking than you have been doing. Remember that you are an educated woman, tuned and timed to distinction and differences. You must cut the telephone wires between your body and your consciousness.

"2. Don't expect too much too fast. One never realizes how sick he was. You were very sick. We are in the summer and you are doing well.

"However, I found myself slipping before this hot spell set in. My stomach and bowels run, with much gas in intestines, side and around heart, with throat and mouth sore, the bowels looser, one day four stools of pale yellow, spongy consistency, at which I left off yeast, frequency now diminished but texture the same, headache, shifting from front to rear of head, old feeling of swelling under left eye and right foot, at times over whole body, occasional little pimples on various parts of body, constant blisters and itching on toes, occasional bright spots in cheek, especially left side, some firing of limbs up to knees. The complexion, which has been clear is darker and cloudy looking with dark circles under eyes. And I do not sleep well at nights.

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The feet could neither be extended nor flexed completely and were apt to remain in the position in which the examiner placed them. He wore tennis shoes because the weight of ordinary shoes was unbearable. The indurated marble like skin extended from toes to mid thigh, from fingers to mid arm, and involved the neck and face.



Fig 228 —Note the glaring eyes, the fixed expression, and the smooth set face. The pigmentation of the hands and feet is plain. The white nails stand out in contrast. (The dark spots are errors in development.)

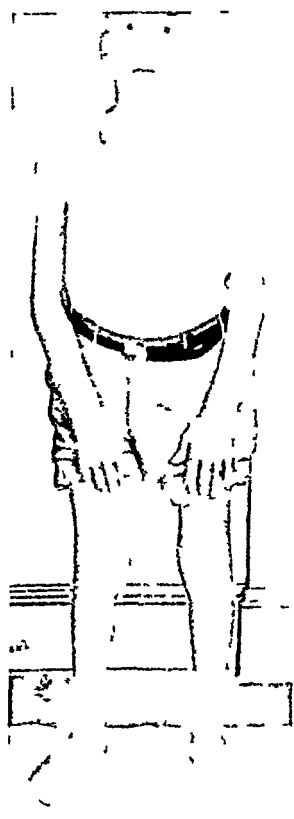


Fig 229 —Note the expression of weakness, emaciation, and the short lips. The excessive pigmentation of the hand and forearm is in marked contrast with the arms and shoulders.

The strength had decreased 75 per cent. The dynamometer reading was only 20—70 to 100 is normal strength. He was very weak and the metabolism test exhausted him. The pulse was fast, 148 after a few steps, and

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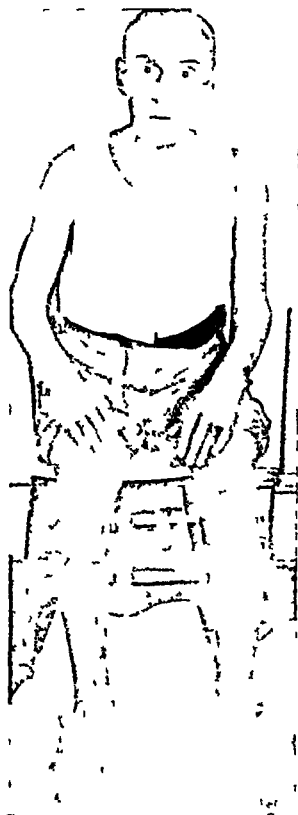


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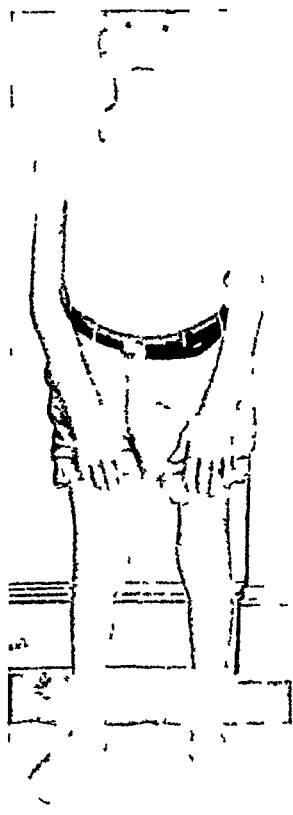


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September 11, 1928 he consulted a physician in Baltimore who confirmed the diagnosis of scleroderma and noted that the disease had extended to an involvement of practically the entire body the more extensive changes were in the extremities. At that time the hemoglobin had dropped to 71 per cent and the white and differential count were normal as was the blood chemistry, stool, urine, and phthalein excretion. The gastric analysis showed a free acid of 5 per cent and a total acid of 24 per cent. Sugar tolerance test showed fasting 0.09 per cent, half hour 0.133 per cent, one hour 0.144 per cent, two hours 0.140 per cent, three hours 0.103 per cent, which would indicate some difficulty in normal glucose metabolism.

A blood-sugar was done March 28, 1928 and found to be 0.11 of 1 per cent and chlorids 550. In April, 1928 he presented the typical picture of erythromelalgia except for an absence of pain. Instead of red and painful feet there were simply the red feet and legs. There was no scleroderma demonstrable at this time. In June, 1928 there was an edema of the feet and legs and hands and marked induration, with subnormal temperature, apathy, and sleepiness so that he would sleep for twelve to fifteen hours at night and repeatedly during the day. A diagnosis of myxedema was made by the attending physician, thyroid extract was administered and there was marked improvement in the induration and edema with decreased sleep and increased mental and physical activity. Treatment was merely palliative. He grew gradually weaker, developed influenza December, 1928, an anuria, had three uremic convulsions with death at 11 P. M. the same day. There was no autopsy.

Osler in his article on "Diffuse Scleroderma" in his *Modern Medicine* discusses the great difficulty of diagnosis in the early stages. The disease may begin with an edema with very slight erythema or as in this case, with very marked erythema and very slight edema. It has been mistaken, as Osler stated, for an early leprosy. April 2, 1928 two opinions were expressed, (a) one that it was a typical erythromelalgia and (b) another that it was a case of circulatory asthenia. In June there was much evidence that it was a case of myxedema though no metabolic reading could be done at that time. August, two months later, the marked

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symptoms of short duration, frequently leaving the heart without definite signs of any structural change or evidence of heart-failure. The symptoms arising from coronary occlusion, although often of the same general character as those of angina pectoris, may be very severe and of longer duration, and repeated attacks are not usually precipitated by physical or mental exertion. The symptoms of coronary occlusion are usually accompanied by a moderate increase in the leukocyte count, and a slight elevation of temperature may also occur. The detection of a pericardial friction rub sometimes furnishes evidence that cardiac infarction has occurred, and symptoms of circulatory failure generally follow occlusion of a main branch of a coronary artery. On the other hand with sufficient clinical evidence to justify the diagnosis of coronary occlusion, apparently complete recovery may ensue, although once the diagnosis is made, the patient must be always thereafter considered as having suffered cardiac damage demanding strict limitation of activity.

The importance of limiting activity after coronary occlusion was strikingly demonstrated by a famous violinist who collapsed on the stage during a concert, and who then came under my care. The symptoms were typical of coronary occlusion, with intense epigastric pain and greatly disturbed cardiac action. The patient made a satisfactory recovery, but showed some slight evidences of heart-failure, and great cardiac enlargement. The warning was given that the tour would have to be cancelled, and the giving of concerts abandoned. The advice, however, was not followed and sudden death occurred immediately before the first concert that was attempted, three months after the original coronary occlusion.

A feature of coronary occlusion that has aroused interest, and is of diagnostic importance is the disturbance of the cardiac mechanism that may follow the sudden cutting off of the blood-supply to part of the myocardium. A series of cases were reported by Robinson and Herrmann<sup>2</sup> several years ago in which ventricular tachycardia apparently followed coronary occlusion. The relationship was not proved in all of the 4 cases reported, as an autopsy was performed in only 1 case. This type of disturbed

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His skin was clear. The pupils reacted well to both light and accommodation. There were no cranial nerve abnormalities and no roughness of long bones. The teeth were good. The trachea was in the midline and there was no tracheal tugging. The chest moved fairly well (vital capacity 3200 c.c.) Slight dullness was found at the right apex but otherwise the lungs were clear. The heart measured by percussion 4.5 by 10 cm., with retrosternal dullness of 7.5 cm. The maximum impulse was in the fifth space and definitely thrusting. The aortic second sound had a liquid musical quality. A systolic murmur was heard over the aortic area and a diastolic murmur was heard at the aortic area, at the left sternal border, and at the apex. This murmur was soft, rather high pitched, and began with the second sound. The blood pressure was 120/60, and the pulse collapsing. The neck veins were not distended. His abdomen was soft, the liver barely palpable, and the spleen not felt. There was no general glandular enlargement. The reflexes were symmetrical and normal.

The urine gave a faint sugar reaction on one examination, but contained no albumin or casts.

Blood. Red blood-cells 5,900,000, white blood-cells 10,700 (73 per cent polymorphonuclears), hemoglobin 94 per cent, Wassermann reaction positive.

The report of the teleroentgenogram was as follows. Greatest diameter of the arch is 7.5 cm. M. L. is 9.5 cm. M. R. is 3.3 cm. Longest diameter of the heart is 15.6 cm. Diameter of the base is 12.2 cm. Greatest diameter of the chest is 27.5 cm.

Two days after admission the patient was seen during one of his attacks of pain. Respirations were 28, pulse 101, blood-pressure 175/60. The patient was slightly pale.

Four days after admission, about thirty minutes after his midday meal, the patient had an attack of pain and dyspnea. He complained of a sense of suffocation more than of pain, and localized this sensation deep in the lower portion of his neck and the upper portion of his mediastinum. There was soreness over the sternum. The face was bathed in cold sweat and the complexion changed to a livid hue. The patient was restless and changed his position constantly. Morphia was given with only slight relief. An hour later the patient was nauseated and vomited, and his earlier symptoms reappeared. Nitroglycerin was given with temporary relief. During the afternoon the symptoms became exaggerated. The pulse became very rapid and poor in quality. He vomited from time to time and complained of pain and a sense of oppression so severe that he actually writhed in his bed. At 6.30 p. m. he began to suffer paroxysmal abdominal pain, associated with involuntary muscular spasm. At 7.45 he was cold, pallid, and slightly cyanotic. The radial pulse was imperceptible. The heart rate as determined at the apex was very rapid, apparently about 160. Twenty minutes later his pulse became extremely irregular for a few moments, but seemed to be stronger. Respirations were shallow and periodic. Shortly before this he became unconscious, having retained his faculties up to that time. At 9 o'clock he was comatose, his head was drawn back, his skin mottled. There was Cheyne-Stokes respiration with apneic periods of about forty-five seconds, during which there were ineffectual gasps. Occasional coarse râles were

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longitudinal crinkling and irregular scarring which appeared to involve both the media and adventitia as well as the intima. These lesions were most prominent where the fusiform dilatations were seen, and they were intimately intermingled with the arteriosclerotic lesions. Here the aortic wall was thinner than normal and on section was definitely scarred. In one place in the descending portion of the arch there was a small outpocketing of the wall which measured 0.8 x 0.4 cm. in size, and which was about 0.2 cm. deep. Its edges were wrinkled and irregular."

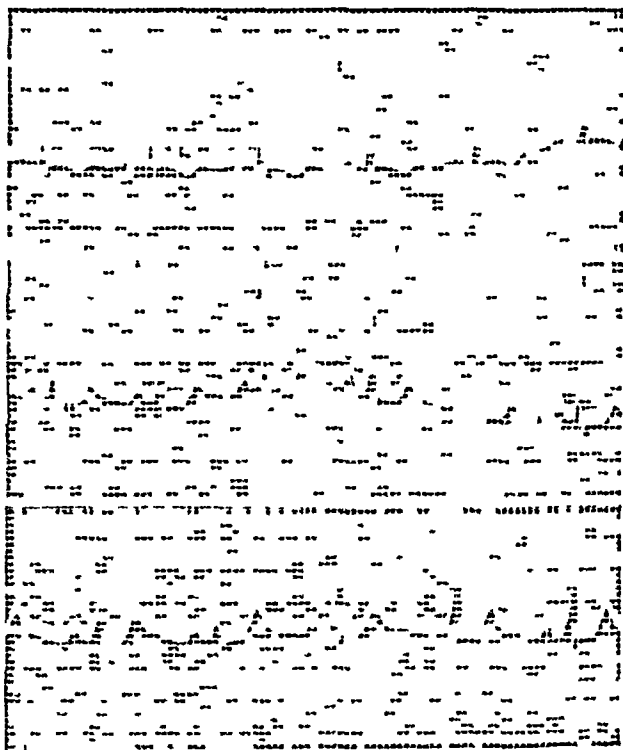


Fig. 231—Record obtained at time of entrance. Leads I, II, and III. Normal rhythm.

**Comments**—This case is reported as an example of coronary occlusion which led to myocardial necrosis, and to ventricular paroxysmal tachycardia. It illustrates the disturbed cardiac mechanism, as revealed by electrocardiograms, which may occur with severe damage of the myocardium. Paroxysmal tachycardia arising from an ectopic focus of stimulus formation is graver from

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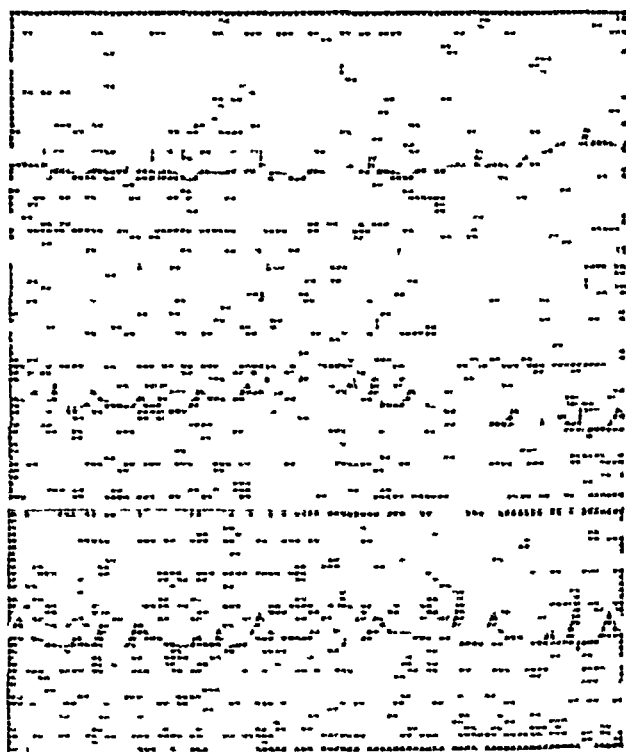


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The case that is reported is of interest from the clinical point of view of distinguishing coronary occlusion from true angina pectoris. The history in this case is in keeping with the latter diagnosis, while the physical examination revealed evidence that would lead to the belief that the coronary orifices were probably distorted, narrowed, and partially closed. The case presents a combination of symptoms and lesions that would favor the belief that some cases of angina pectoris are caused by lack of blood-supply to the myocardium due to a lesion of the coronary vessels. Such lesions are, however, lacking in many fatal cases of angina pectoris.

I wish to express my thanks to Dr C Sidney Burwell for his collaboration in preparing this report.

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several severe attacks of epistaxis and on two occasions there was oozing of blood from the gums. For four days before admission the left foot and leg were swollen. There was no pain and no disturbance of sensation at any time. The boy worked at a lathe in a machine shop until the day before he came to the hospital.

Physical examination showed a boy of good bony and muscular development, but of distinctly abnormal conformation. The temperature was normal, the pulse 88 per minute, the respirations 20 per minute, and the blood pressure 120/75. The facial expression was peculiar, the mouth held open and the lips and face drawn in a fixed insane grin. Speech was hesitant with slurring and at times mumbling of words, almost every sentence was prefaced by a high-pitched laugh.

The bodily conformation was eunuchoid with some feminine characteristics. The arms and legs were disproportionately long and were rounded and tapering, the fingers in spite of work as a mechanic were slender and delicate. There was complete absence of axillary and pubic hair. The mammae were of feminine type, the nipples large with a wide areola and with glandular and fatty development comparable to that of an eleven-year-old girl. The pelvis was wide, the mons large, the genitalia minute, the thighs and buttocks typically feminine.

The head was anatomically normal, the ears and sinuses showed nothing of significance. The eyes looked normal, extra ocular movements were well performed, there was no nystagmus. The pupils were normal in size and contour and reacted to light and distance. The fundi showed no abnormalities of nerve head, vessels or retinae. The nose was negative. The teeth were well preserved though there was pyorrhea, the tongue was protruded in the midline without hesitation or tremor. The tonsils were large with no gross infection, there was no salivation and no dysphagia. The neck showed no abnormality.

The thorax aside from the exuberant mammary development was quite negative. The lungs and heart showed nothing noteworthy. The abdomen was smooth and rounded, the left hypochondrium bulged, respiratory movements were good. The edge of the liver was felt one fingerbreadth below the right costal margin, the spleen reached three fingerbreadths below the left costal margin and extended almost to the midline, the edge was thick and rounded, very firm. No signs of free peritoneal fluid were elicited. The genitalia were very small, comparable in size to those of a six-year-old boy, both testes were present in the scrotum.

The arms were flexed at the elbows and held crossed on the thorax, the hands were flexed on the wrists and the fingers flexed on the hands but the fingers themselves were held in extension and slightly separated. This flexion spasm could be overcome slowly by the patient and readily by the examiner. There was no tremor, athetosis or fibrillation, the muscles were not atrophied, the grip was excellent and the strength of the arms surprising. There was no definite inco-ordination, but movements were performed very slowly. The legs were held flexed as the patient lay in bed with knees drawn up and feet extended on the ankles, movements were well performed and the strength of the legs was good. The boy stood with his knees slightly

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life of the patient " Here we have a youthful patient who has developed successively spasticity, with curious and characteristic posture of the face, hands, and feet, dysarthria, tremor, dysphagia, emotionalism, and emaciation There is no true paralysis, no real contracture, the reflexes remain qualitatively normal In addition to these neurologic findings we have a history of nosebleed and oozing from the gums, there is hepatomegaly, splenomegaly, and recently, ascites, all pointing to hepatic cirrhosis Our patient presents in addition, evidence of gonadal insufficiency

The etiologic agent in this disease remains unknown Wilson found it often a familial disease No other member of our patient's family as yet presents evidence of the malady It is the consensus of opinion that the hepatic disease is primary with secondary toxic degeneration of the lenticular nuclei It is curious that symptoms of cirrhosis of the liver should be so infrequent clinically when profound changes are uniformly present postmortem Our patient is, so far as I have been able to discover, the first to present definite evidence of hepatic cirrhosis during the course of the syndrome of progressive lenticular degeneration

*Further Course and Autopsy Report*—The progress of the disease was rapid On December 1st slight icterus was noted, the patient lost weight and strength rapidly Dysphagia became complete so that tube feeding was resorted to on December 10th The mental condition became much worse At night he was delirious, during the day he was drowsy and could be roused only with difficulty He became incontinent, or was unable to indicate when the necessity for voiding and defecation arose The ascites and edema increased steadily and the daily elevation of temperature reached 101° F, a severe secondary anemia developed On December 28th small decubitus ulcers appeared over the ischial tuberosities, these rapidly increased in size On January 20, 1924 the temperature rose to 102° F, signs of bronchopneumonia were found The patient died of bronchopneumonia on January 24th An autopsy was performed by Dr E R Pond

*Autopsy Findings*—(Autopsy done ten hours after death) The body is that of an emaciated white male about eighteen years of age, 177 cm long, 40 kg in weight Good bony but poor muscular development Postmortem lividity in dependent portions Two large, deep, sloughing, ulcerated areas over the ischial regions Rigor absent No axillary hair present and very few pubic hairs

Cranial hair rather thin Eyes deeply sunken in orbital fossæ, pupils equal, regular, half contracted, scleræ slightly yellow Nose clean Sordes on lips, teeth, and tongue Teeth in good state of preservation Neck long and thin Thorax, long and narrow Mammary are prominent as soft elevated areas just beneath and around the nipples about 5 cm in diameter, the left slightly larger than the right Abdomen distended Extremities show nothing unusual

Scalp thin and rather dry Calvarium of the usual thickness Dura translucent Sinuses filled with fluid blood Pia is transparent and free from the dura except along longitudinal sinus Cerebrum symmetrical Convulsions appear somewhat flattened and are not as prominent as usual The brain is diminished in consistence generally, probably postmortem

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Ureters Nothing unusual

Bladder Firmly contracted, contains a small quantity of clear urine

Genitalia Penis small Testicles quite small and firmer than normal

Prostate shows nothing unusual

Gastro-intestinal tract Peyer's patches are readily visible as flat, well defined purplish areas Mesenteric lymph-nodes are from 1 to 2 cm in diameter and purplish in color

Pancreas Anterior border green stained, consistence diminished On section the lobules separate more readily than normal

Anatomic diagnosis Bronchopneumonia, bilateral lenticular degeneration, cirrhosis of liver, splenomegaly, ascites, emaciation, hyperplasia of breasts, delayed puberty, jaundice

Microscopic examinations

Cerebral cortex Nothing unusual

Right lenticular nucleus Region of putamen, marked gliosis and a few areas of degeneration No ganglion cells seen Region of the globus, few areas of gliosis and slight degeneration, some pigment present

Left lenticular nucleus Region of putamen, extreme gliosis with marked degeneration, small amount of pigment present throughout, no ganglion cells seen Region of the globus, marked gliosis with areas of degeneration, yellowish pigment scattered throughout, no ganglion cells seen

Left thalamus Marked gliosis and much degeneration

Cerebellum and medulla show nothing unusual

Pituitary Much colloid in the intermediate portion There are focal areas of necrosis in the pars anterior

Lungs The bronchi and alveoli contain varying amounts of blood, serum, fibrin, and polymorphonuclear leukocytes

Heart The fibers are small, there are a number of large colonies of bacteria

Liver The parenchyma is divided into irregular islands by broad bands of dense connective tissue In some areas this connective tissue is infiltrated with lymphocytes and a few polymorphonuclear leukocytes and the bile-ducts are increased in number The liver cells are in various stages of necrobiosis Scattered throughout are colonies of bacteria

Spleen Intense congestion The lymph-nodules are few in number and very small The pulp is markedly diminished in amount The endothelium of the sinuses is heavily pigmented There are large focal areas of necrosis, also occasional colonies of bacteria In other sections where the congestion is not so intense there is an increase in the amount of connective tissue about the sinuses

Adrenals In the reticular zone of the cortex there are numerous large colonies of bacteria with necrosis of much of this area

Kidney The capsule is regular Of the glomeruli there is nothing unusual The tubular epithelium for the most part is cloudy and swollen There is marked congestion of all the vessels

Urinary bladder Slight lymphocytic infiltration of the mucosa

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prolonged, soft, blowing, systolic murmur audible at the mitral and pulmonary areas, otherwise the heart sounds are normal. The heart rate varies from 92 to 104 per minute, there is a marked sinus arrhythmia. The blood pressure is 90 systolic, 60 diastolic.

The abdomen is rounded and bulges slightly in the right hypochondrium, it feels soft, there is no tenderness anywhere. The edge of the liver is felt 3 cm. below the right costal margin, firm, smooth, and rounded. The spleen cannot be felt and no area of splenic dulness can be percussed. No masses can be felt and there is no dulness in the flanks. There are a few scattered pubic hairs. The external genitalia are infantile.

The right leg shows a round grayish scar the size of a quarter over the tibia, 2 inches above the malleolus. The left leg presents a shallow, granulating ulcer approximately  $1\frac{1}{2}$  inches in diameter over the midportion of the middle third of the tibia, there are two small scars of healed ulcers between this and the ankle.

The blood is most interesting. On admission the hemoglobin was 44 per cent, the red blood-cells 2,468,000, the white blood cells 11,200. A stained smear showed the characteristic poikilocytes which we call "sickle cells" elongated fusiform, banana-shaped, and crescentic erythrocytes which stain deeply and uniformly with Wright's stain. These made up 26 per cent of the total number of red blood cells. The red cell picture otherwise was that of a severe secondary anemia, with anisocytosis, ordinary poikilocytosis, marked central pallor of the cells, diffuse and punctate basophilia and occasional normoblasts. A smear stained with brilliant cresyl blue showed 18 per cent reticulocytes. The leukocyte count was somewhat lower than is usual in this condition. Leukocytosis up to 60,000 without demonstrable infection may occur. A differential count of 500 leukocytes showed 63 per cent polymorphonuclear neutrophils, 16 per cent eosinophils, 06 per cent basophils 78 per cent large lymphocytes, 234 per cent small lymphocytes, 14 per cent monocytes, 1 per cent of damaged cells. Four normoblasts were seen while counting. Malarial parasites were not seen. The platelet count was 280,000. A fragility test showed that hemolysis began at a concentration of 0.45 per cent NaCl, and was complete at 0.24 per cent. The van den Bergh reaction was strongly positive in the indirect phase. The Wassermann was negative. The blood sugar was 105 mg per 100 cc and the non protein nitrogen 24 mg per 100 cc. In a wet preparation sealed and allowed to stand at room temperature for twelve hours it was found that all the erythrocytes had assumed "sickle" shapes and that many of the leukocytes had phagocytosed erythrocytes. Numerous examinations of the blood since admission have showed no material change in the picture.

The urine was amber colored, acid and clear, the specific gravity was 1.012, albumin sugar and bile were absent, urobilin was present in large amounts. A few granular casts were found on microscopic examination. The phenol sulphonephthalein test showed a total two hour excretion of 48 per cent.

**Discussion** —This girl presents all the typical findings of a peculiar blood-dyscrasia which is called "sickle-cell anemia

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The morphologic changes in the blood in the active phase are twofold, those which are characteristic of the disease and those which are common to secondary anemias. The poikilocytes which give the disease its name are seen in no other condition and no similar deformity has been produced experimentally. Sick cells are found in relatively early stages of development in the bone-marrow, and though 100 per cent of an "active" patient's cells may become "sickles" in wet preparations, the bizarre forms which result from the sickling of round cells never look exactly like the sickle cells of fresh blood or marrow preparations. The circulating sickle cell has a concentration of color and high degree of refractility that are all its own. In marrow preparations numerous nucleated sickle cells are seen and not infrequently they are found in blood smears. The anisocytosis, basophilia, large number of reticulocytes, and normoblasts and perhaps the increase of leukocytes are manifestations of bone-marrow activity such as are seen in any severe secondary anemia.

The blood-platelets seldom vary beyond normal limits. The fragility of the erythrocytes is not increased. Mason<sup>4</sup> observed that the cell residue in the lower concentrations of salt solutions as complete hemolysis was approached consisted entirely of

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endothelial system In the liver there is evidence of marked phagocytic activity on the part of the Kupffer cells The spleens in our active cases have been small, with great fibrosis, partial obliteration of the lymphoid nodules, and great distention of the sinuses with sickle cells There is abundant pigment deposition in the spleen in the endothelium of the sinuses, in the perivascular connective tissue, and in free macrophages In 2 cases numerous hemorrhages had occurred into the splenic pulp The bone-marrow is hyperplastic, numerous nucleated sickle cells are present, and there is abundant pigment

The treatment of this condition is most unsatisfactory We felt that by analogy with familial hemolytic icterus, the hemolysis in this disease might be controlled by splenectomy By some irony of fate we have never been able to secure consent for the operation on one of our cases In the instances elsewhere where the operation has been performed<sup>2,13,14</sup> there seems to have been relief of anemia, but no effect on the morphology of the blood It is probable that splenectomy is most effective during the splenomegalic stage of the disease Liver diet has been tried, in fact, this patient has been on this diet since admission We have seen no effect from it Transfusions may serve to tide a severely anemic patient over a critical period, but have no lasting effect

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this district was of English, Scotch, Welsh, and Irish descent. These men were brought up in the mines from childhood and knew nothing of any other means of making a living, and preferred working in coal mines to any other method of employment, but the sons of these men have shown no inclination to take up their father's type of work. So far as the Tennessee Coal, Iron & Railroad Company is concerned, the white miner has almost disappeared.

Owing to the isolated position of many of our communities, such as have been described, it became evident many years ago that some method of medical attention would have to be devised for the workers and families who lived in these places. To supply this need contracts were made with physicians on the basis of so much per man per month. This ranged from \$1 00 to \$2 00, and was cut out of the employee's wages and given to the physician in a lump sum.

The communities being of considerable size, and in those days health conditions being not what they might have been, a great deal of medical work was necessary. Those physicians that held the original contracts with the company employed men under them at a fixed salary to carry on the medical work under the direction of their immediate chief. Small hospitals were erected by the company to take care of accident work, injuries, minor surgical operations, and occasionally major operations when such seemed necessary. The entire work of the Tennessee Coal, Iron & Railroad Company was divided into some five or six contracts, and entailed before the changes which are to be described took place in the medical department, the employment probably of some 18 or 20 doctors.

It must have been apparent to the most casual observer that the development in the Medical Department and its relation to the employees of the Tennessee Coal, Iron & Railroad Company had not kept pace with the development of other departments. The Tennessee Coal, Iron & Railroad Company at that time was just entering into a period during which the employees, their comfort, their homes, and their general treatment were requiring a great deal more attention than had ever previously been

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This method of work of course was very unsatisfactory, although considered a vast improvement over the work which had been previously done

The period of 1913 to 1918 was one of organization, development of an efficient sanitary department, and eradication of communicable diseases

In November, 1919 the Employees' Hospital was opened in Fairfield This hospital, of modern construction in every way, has a capacity of some 300 patients The staff was selected from our own physicians, with the exception of a few specialists This staff has been added to since the hospital was opened, and such advancement as has been made in position has been confined to our own people, that is to say, no new blood has been brought into either the medical or surgical department, except that which we have ourselves developed

The hospital was opened at a time when x-ray therapy was just beginning, when the improvement of x-ray machines was in its infancy This department has grown beyond all expectations, occupying at the present time at least four or five times the room originally set apart for it The physiotherapy department, of which we had not even heard, when the hospital opened, now occupies a section in the hospital almost comparable to that occupied by the x-ray department

Radium has been added, the best type of deep x-ray therapy, and every form of physiotherapy, with the exception of hydrotherapy The hospital contains at the present time as complete an equipment as is likely, I think, to be found in any hospital in America

In the nine years which have elapsed since the hospital opened, we have developed men to handle the traumatic eye work, the genito-urinary work, obstetrical work, ear, nose, and throat work, and pediatric work The amount of work which passes through this hospital is capable of developing in individuals, even of most ordinary intelligence, a very considerable degree of skill

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It is very likely that the men working on a small salary, caring for large numbers of people with very scant equipment, with no hospital facilities, with few if any laboratory facilities, could give them, even if the doctor did his very best, but a low grade of medical service. From what I can gather in this district, that is what they did give, and that is the only thing they were able to give with the disadvantages under which they worked.

It took quite a while for us to convince many of our employees, and the convincing I think was done simply by deeds, which proved that we were anxious and able to give to them a quality of medical and surgical attention which compared favorably with anything they could receive elsewhere. Given the facilities with which we now work, the hospital, its remarkable equipment, and its all-time staff, we would be very dull, to say the least, if we could not give this type of attention.

It has been a slow process convincing some, but the some have come to be but a few now, and every year we have had more and more converts. It is true that there are still some people among our employees, very possibly a considerable fraction, who come to us for the sole reason that it costs them less than to go outside. But as time has gone on we are having a very high percentage of readmissions, the people returning to the hospital not grudgingly because it is cheaper, but willingly because they have become convinced that they get here the best return for their money. In certain of our settlements the percentage of men who have gone willingly on the medical fee list, that is, paying in a certain amount every month, has reached 100 per cent, and has shown every sign of remaining at that figure.

Opposition from the other group of individuals, our colleagues in the medical profession, has been a much more serious matter. As long as we confined ourselves to Out Clinic work in the small dispensaries in the various districts and in the operation of two small hospitals, we heard little, if any, criticism of our work, and none at all in any organized way in the State and County Medical Society.

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What were these ideas, and how did they intend to enforce them? Probably the best way to describe what they had in view is to insert a copy of resolutions which were presented before the Jefferson County Medical Society in the Fall of 1925. They were adopted by a working majority, and were carried down and presented for the consideration of the Alabama State Medical Society in Mobile in the following year:

"Whereas, for a number of years in Jefferson County flagrant and gross violations of the code of ethics of the American Medical Association as well as of the ordinance of the State Medical Association regulating Contract Practice in this State have been known and have been practically ignored by the Jefferson County Medical Society

"And whereas, all efforts in the past to suppress the growing evil in the profession have failed

"And whereas, these violations have increased each year to such an extent that the medical profession of Jefferson County has become the object of severe criticism and censure by the profession throughout the State of Alabama and other states

"And whereas, the high esteem and confidence in which the medical profession has always been held by the public is gradually but surely being lost

"And whereas, these violations of medical ethics constitute a very grave offense and tend to lower the tone and to destroy the cherished traditions and high ideals of an honorable profession, it becomes imperative that some drastic measures be adopted to arrest this growing menace

*"Therefore be it Resolved*

"First That all list contracts of any character made by any hospital or physician or groups of physicians, mutual aid associations or Insurance Companies of any character providing for medical or surgical services upon the payment of a daily, weekly, monthly, or yearly stipend be declared unethical

'Second That it be declared unfair competition and contrary to the ethics of the American Medical Association for any corporation or company who maintains its own hospital to take any patient other than its own employees and their immediate families for hospital medical or surgical services except accident cases occurring in the vicinity of the hospital

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be instructed to forward a copy of these resolutions to the State Board of Censors and to the State Committee on Contract

"Thirteenth That the Board of Censors be requested to take these resolutions under careful attention and consideration and to render a report at the next meeting of the Jefferson County Medical Society, October 23, 1925 "

Let us review this resolution without prejudice I think that all the statements made in the preamble may be accepted at their face value So far as the resolutions themselves were concerned, it might be said that they took up the question of what is and what is not ethical practice to a degree and to an extent which, so far as I know, has never been taken up before

In the second paragraph of the resolution a definite effort is made to limit the work of any hospital, but in this instance it referred to the Employees Hospital of the Tennessee Coal, Iron & Railroad Company, to the treatment of employees and their own immediate families, with the exception of accident cases Regardless of the rights or wrongs in the matter, and regarding it in a purely judicial way, this is the first attempt of which I am aware that has been made to limit the usefulness of any hospital in this country in such a way

In the third paragraph of the resolution any physician employed by a company is limited in his practice solely to the care of the employees of the company for which he is working The ninth and eleventh articles of the resolution combined form together the punitive part of the document As is well known, it is difficult or impossible for physicians who are not in good standing with the County Medical Society to obtain liability insurance for their practice So far as the Health Department of the Tennessee Coal, Iron & Railroad Company is concerned, the meat in this cocoanut is confined entirely to two items in the resolution, the first being the effort to restrict the hospital solely to the use of the employees and their immediate families, and the second item that which made it obligatory for any physician employed by a corporation on a whole time basis to confine his attention entirely to the service of the employees and their families

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One of the proponents of the original resolutions, which have been quoted before, who was a member of the State Board of Censors, handed in a minority report in which he stated that he could not concur in the report of the majority. This minority report was taken up first in the voting, and was defeated by a vote, which included the counsellors and delegates of the Medical Association of the State of Alabama, of 80 to 75. After a short discussion the majority report of the State Board of Censors, the gist of which has been given above, was adopted unanimously. Thus was the matter settled, insofar as it can be settled with two large factions in the medical society possessing such divergent lines of thought.

In 1913, when Dr. Noland had his first conference with Dr. Sanders, then the State Health Officer, a gentleman's agreement was entered into by which the monthly stipend for medical service would be collected from the employees of the Tennessee Coal, Iron & Railroad Company who earned less than \$250 per month. This has been adhered to.

It might be said that at the meeting in 1928 the following recommendation regarding this troublesome matter was made by the State Board of Censors, and was unanimously adopted. This is the last official statement concerning this matter by the State Medical Association.

"That the term 'unofficial employee' means any employee, who is not financially able to obtain efficient medical and surgical attention in times of sickness and distress. The employee's income under this interpretation will vary in different parts of the State according to the economics and living conditions in the community in which the particular contract is entered. It is assumed that each country medical society shall have authority to determine the income limit for the contracts within its boundaries, as provided for under the terms of this interpretation. For instance, \$150 per month in some communities would be equitable, \$200 per month in other communities would be equitable, but in no case, and under no circumstances, and in no locality, shall the income of the employee claiming benefits under

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number of men engaged in work of this sort in all probability will grow. They, being thoroughly satisfied with the character of medical work which they perform, will of course fight for their position in the County and State Medical Society, feeling that they have done nothing which should cause them to lose such rights.

Judging from the character of the resolutions which were proposed, and which narrowly escaped passing, certain members of the Medical Society believe they have the right to deprive one of his position as a member of the County Medical Society if he does not conform to their idea of what medical ethics should be. It is undoubtedly true that for various offenses a man may be expelled from a County Medical Society, but on scrutiny these offenses would be found to be offenses primarily against the law of the land or laws of human conduct which would be just as reprehensible outside the medical profession as within it.

Efforts of this description to discipline members of the Medical Society solely on their supposed breach of medical ethics is a new project. To transfer contests between different factions of medical societies over strictly ethical matters to the law courts would subject them to a degree of analysis and criticism to which they have not been exposed before.

Here in this State members of County and State Medical Societies constitute the State Board of Health with definite legal rights. That a society on some so-called ethical ground, which did not involve the question of moral turpitude, could deprive any member of such rights is considered rather doubtful. As pointed out by the late State Health Officer during the argument in the State Medical meeting at Mobile in 1926, if these resolutions had passed in all probability the next step would have been to the courts. Litigation of this sort is prolonged, expensive, and so far as State and County Health and Medical Associations are concerned in the end would I believe, be ruinous. Just what compromise can be worked out remains to be seen.

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They regarded this development as an opening wedge for projects of a similar character, and they felt that in this proceeding members of the profession would frequently be exploited to their own undoing for selfish ends. I think they were perfectly correct in this view. It is exactly what is going to happen, and no doubt has happened. I believe developments along this line will be rapid, and in a decade or so will be fairly widespread. They will be good, bad, and indifferent, just as any medical service today is good, bad, and indifferent. I believe that in certain instances doctors will be overworked, underpaid, and various other forms of injustice will be apparent and easily proved, but I do not believe that such systems of medical care will long survive, if conducted along these lines. In all likelihood the introduction of these methods will be largely through corporations, but there is no question in my mind that the time will come, and shortly, when communities will obtain, if they so desire, group service in a well regulated hospital from competent men at a rate which all can afford.

I think it is only fair to say that the Medical Profession as a whole appears to be opposed to any innovation which threatens their individual independence, no matter how valuable the change may be to the community or to a group consisting of large numbers in any community.

Little criticism of the medical profession becomes articulate, but we know it exists, and one indication is the rapid growth of many cults and isms. Dr M L Harris, writing in the Journal of the American Medical Association, November 26, 1927, states that in the last several examinations for licensure in the state of Illinois the number of chiropractors, osteopaths, etc. exceeded that of the regular profession. He remarks that it is not unreasonable to suppose that there must be some demand on the part of the public for the services of cultists, otherwise there would not be such a large number desiring to enter their ranks.

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I believe that multiplying instances of this description have counted more for the lack of confidence in us, which we find so widely scattered throughout the country, than has any other reason. Hampered by the peculiar ethics of our profession in our silence we connive at this really appalling state of affairs.

The medical profession, I think, has far more than its fair share of rascals. Any profession which is so robed in mystery, so surrounded by all sorts of subterfuges which appeal to those of devious and furtive mind, would inevitably attract and hold individuals of this type.

It has been unfortunate for the medical profession that it is regarded by tradition as a sort of priesthood, working on a plane far above that of the butcher, the baker, the candlestick maker. Removed from the desire to acquire. Above temptation to do anything except that which made the medical tradition what it is. This tradition, as we know, bears very little relation to the facts. Doctors are just like other human beings. They have the same ambitions, the same tastes for luxuries, and the same desire in most instances to meet their ever increasing financial obligations with promptness and honesty.

The old time family practitioner is often held up as a sort of an ideal. The type of man who never sent bills, or sent them only annually. Such an individual would make a pitiful figure in the world as we know it today, and would inevitably end up in the court of bankruptcy.

Those graduating from schools of medicine at the present time very quickly shunt themselves into fields of activity where the return for their work will be prompt and generous. They certainly cannot be blamed for this. We are attempting the very difficult task in our profession of pursuing an altruistic and unselfish ideal in one direction, while busily employed in trying to make as much money as we can by moving sometimes in the opposite way. That is one thing that has made the whole matter so trying, and has cast a semblance of hypocrisy over our acts which, on the whole, I believe is an injustice.

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early as their more hard-worked sisters. The more fortunate ones, who have kept their youthful appearance and vigor, as a general rule, will be found to be the ones who have been better fed than the average.

So widespread is the existence of malnutrition that it occurs to the examiner as a possible, even though remote etiologic factor, in almost every patient seen regardless of the actual pathologic condition found. It looms up in the background of practically every case, and we soon learn to study each patient with its possibility in mind. In addition to vitamin deficiency, the examiner often senses a possible mineral deficiency, even though this may be difficult to prove. At times one feels that iodine, arsenic, iron, calcium, or perhaps phosphorus may be lacking, for frequently the diet is deficient in more than one important element.

One of the most difficult things confronting the physician is to find out just what his patients eat. They will tell him with reasonable accuracy the menus served on their tables, but they are extremely vague as to what they themselves actually consume. It is only in comparatively recent years that we have begun to study dietary habits, but now that we are beginning to learn the harmful results of an unbalanced or deficient diet it is important to know in detail the food habits of the patients with whom we deal.

The observations recorded in this paper are the result of several years study of this question in central Alabama. The patients studied were drawn in about equal proportion from rural and urban population. By far the greatest dietetic difficulties are met with in the rural population. In the cities, where access to a good market is possible at all times of the year, our problem is much simpler, but in the country the problem of a proper diet is at times almost insurmountable.

In Alabama and, probably in the other Southern States, there are comparatively few all the year gardens, in spite of the fact that fresh vegetables of some kind can be grown the year round. Reliable authorities estimate that only 50 per cent of the farm families of the state have an adequate milk supply, and that

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and insufficient ration is not universal, but it is altogether too prevalent and prevails, perhaps, in the majority of rural homes. The caloric value is usually ample, and at times of the year there is a plentiful supply of fresh vegetables, fresh fruit, and fresh milk, but this is woefully inadequate except during early spring and summer.

Dickins, in a study of the food habits of 100 representative families from two different areas of Mississippi, summarizes practically as follows: (1) That scarcely more than was actually necessary of energy, protein and phosphorus was well provided, that calcium was well provided, that iron was not provided in sufficient quantities, that vitamin A and B requirements were probably met, and that vitamin C was low in fall, winter and early spring.

(2) Little seasonal variation in the nutritive values of these Mississippi dietaries was found.

(3) The average number of physical defects per child per family was higher for the families whose dietaries were below standard in two or more nutritional factors.

These conclusions probably accord closely with the average in the Southern States, except that in many instances the supply of calcium is insufficient for the growing child, due to the small amount of milk taken by so many children. In my observation not many children get the quart of milk a day which is supposed to be the minimum amount necessary for adequate growth.

The effect of unbalanced, insufficient diets has not been seriously enough considered by the profession as a whole. Of course, the definite deficiency diseases, such as rickets, scurvy, and possibly pellagra, have been thoroughly studied and their relation to food is well known. But it is extremely likely that there are many "vague more or less indefinite states of malnutrition" and ill health resulting from dietary insufficiency that are not recognized. There is a type of individual seen in the South, usually scrawny, with dry, harsh skin, often underweight, lethargic, complaining constantly of "biliousness" or "malaria," resorting to calomel frequently, going from one doctor to another without relief, who will improve promptly when put on a diet containing green

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ern cooking deserves all its reputation, but, unfortunately, this excellence in the culinary art is confined to the few. It is a distressing thing to see good material ruined in its preparation, yet wretched cooking is a daily occurrence, even in many homes of the well-to-do. Vegetables are greasy, poorly flavored, often cooked too long. The bread is heavy and soggy. Many housewives can only fry and boil meat, and the end product is usually swimming in grease. The chief food dislike among children is for green vegetables and this distaste is acquired very early in life. The explanation, I think, is to be found in the miserable way in which the vegetables are prepared. The vitamin content of many of the leafy vegetables is probably destroyed by the long boiling to which they are so often subjected, so that even during seasons of the year when vegetables are plentiful, there may be a vitamin deficiency just the same.

Several years ago I was consulted by a rural physician, who had such severe digestive trouble that his condition had been diagnosed as gastric cancer. Study of his case revealed clearly that it was one of dietary indiscretion rather than malignancy, and improvement followed institution of a better diet, but even then the symptoms persisted to a certain extent. Several months after I first saw him I had occasion to take a meal in his home and it became apparent at once why his trouble was not cured. He was eating the right things, but they were cooked in the manner just described. The vegetables were greasy, the biscuits heavy and soggy, and a large dish of fried chicken, swimming in grease was the *piece de resistance*. He ate heavily not only of the solid food, but took with the greatest relish spoonful after spoonful of the greasy gravy. After the meal I called him aside, made due apologies for being critical, and showed him the utter impossibility of recovery on such food. Fortunately he realized the justice of the criticism, instituted the necessary changes, his trouble cleared up and has not since recurred.

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the dietary needs of their children and this has helped to make the handling of adults a far less difficult problem than was the case even fifteen years ago

The remedy for our situation is easy to know, but difficult to apply. In the first place we should begin with the profession itself for most of us have paid too little attention to this important phase of our work. The State Boards of Health and the various Extension Departments of our State Schools are doing a great deal of good work in educating the laity, but after all, the individual physician is the man who bears the chief responsibility. He gets closest to his patients and they value his counsel more than that of any one else. Not until he takes a serious enough interest in dietetic problems to advise his patients properly will a great deal of headway be made.

It is not enough to take a diet slip from a text-book and give it to a patient. One must know what foods are available to the particular patient, construct his list, from them, and teach the patient how to use them. Most of our text-books on diets have been written by Northern writers and many foodstuffs in their lists are not available to our Southern people, yet there is no time of the year when all necessary food elements are not obtainable in this section. We should preach to the people the need for year round gardens, and the advisability of raising more of their foodstuffs than they do at present. The Southern diet in the summer should consist almost entirely of milk and milk products, raw and cooked vegetables, raw fruits, bread, and a minimum amount of meat. The necessary protein can be obtained from the milk and a small amount of meat, while all the other necessary factors will be yielded by the vegetables, fruit, and bread. In the winter more meat and eggs, cereals, desserts, and nuts can be added, but even then vegetables, fruit, and milk should constitute the bulk of the food. Our Southern barbecue, delightful as it is, is really a bad institution. The rich camp stew, the barbecued meats are truly delightful to the taste, but they are winter time foods and should not be eaten in the summer, the time of year at which barbecues are generally held.

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